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Haley & Aldrich, Inc.

VOLUME III

HEALTH AND ENVIRONMENTAL RISK ASSESSMENT

PARCEL 18 AND 18B

BOSTON, MASSACHUSETTS

Consulting

Geotechnical Engineers,

Geologists and

Hydrogeologists

[redacted]

VOLUME III

HEALTH AND ENVIRONMENTAL RISK ASSESSMENT

PARCEL 18 AND 18B

BOSTON, MASSACHUSETTS

Prepared For

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EXECUTIVE SUMMARY

This document provides a public health and environmental risk assessment for the Parcel 18 & 18B site near the Ruggles Street MBTA Station in the Roxbury section of Boston, Massachusetts. The assessment relies on field data reported in Volumes I and II, Phase Two Oil and Hazardous Material Site Evaluation prepared by Haley & Aldrich, Inc. The risk assessment: briefly describes the site; describes the risk assessment method required by the Massachusetts Department of Environmental Quality Engineering; identifies the Compounds of Interest, potential exposure routes, receptors, and exposure point concentrations; develops exposure scenarios; and provides a dose response assessment and risk characterization. The appendices include details of the risk characterization and toxicity profiles for Compounds of Interest.

Section 1 includes a site description and rationale for the risk assessment method selection. The site is an urban site consisting of three landscaped and fenced parcels of land. It is directly opposite the MBTA Ruggles Street Station. Current access through the site is a pedestrian walkway to the station. The surrounding community includes commercial and residential development, a local high school, and a nearby park. The proposed use of the site is as office and commercial buildings. The first building scheduled for construction is Tower 2 and an above ground parking garage. Construction will be above ground. The only planned excavation concerns auguring to advance supporting piles, pile cap excavation, and any excavation to accommodate the slab foundation. This first construction phase may be followed by two subsequent construction phases.

Soils and groundwater at the site contain low levels of various chemical contaminants. The soil contaminants occur at depth (4 to 20 feet) and in near surface soils (< 4 feet). Contaminants include volatile compounds in the deep soils; semi-volatiles in surface and deep soils; metals in surface and deep soils; pesticides in surface and deep soils; and pesticides, low levels of volatiles, and metals in groundwater. Section 2 of the report provides a detailed list of contaminants considered in the risk assessment.

The exposure assessment in Section 3 includes details of construction plans, and a description of exposure pathways. The

likely worst case exposed populations include construction workers, pedestrian commuters walking by the site to Ruggles Street Station, children playing in either the nearby (150 meters away) playground or the front areas of the nearby residences (50 meters away), and daycare children playing in a proposed Phase III play area. The exposure points and pathways for these receptors include: exposure to deep soils (dermal contact, incidental ingestion, and inhalation) by construction workers during pile installation and pile cap excavations; exposure to surface soils (incidental ingestion, and inhalation) by construction workers during staging activities for building construction; exposure of pedestrian commuters and children (inhalation) to airborne particles during all phases of construction; and exposure of daycare children from surface soils.

The report estimates average daily dose based upon accepted parameters for soil ingestion, contaminant absorption, and body weight, and estimated averaging period (for threshold effects) and a lifetime exposure over 70 years for incremental risk due to non-threshold effects. We used these parameters to estimate average daily dose from each exposure pathway.

Section 4 is a dose response assessment for the Compounds of Interest. The information in this section includes chemical profiles, cancer potency factors (or relative potencies) for carcinogens, and reference doses (RfD) for non-carcinogenic compounds. The RfD's are also provided for carcinogens with systemic health effects.

Section 5 present the risk characterization for each receptor population for each proposed phase of construction. Contaminant concentrations in soil and groundwater and calculated concentrations in airborne particles were compared to known standards. Some of the groundwater samples exhibited contaminant concentrations in excess of drinking water standards for heptachlor, lindane, and nickel. However, the groundwater in the area is not a water supply. Calculated on-site and off-site air concentrations were orders of magnitude less than standards.

We calculated risk due to carcinogenic and non-carcinogenic exposure for each compound of interest separately. Risk from carcinogens was calculated by multiplying the average daily dose for each carcinogen by its cancer potency factor or its relative potency and summing the ratios over the entire range of Compounds of Interest. Figures ES-1 to ES-3 provide the results of the calculations. None of the carcinogenic risks (total) estimated for any of the receptors exceeded the DEQE the 10^{-5} (one in one-

FIGURE ES-1 CARCINOGENIC RISKS DURING PHASE I CONSTRUCTION

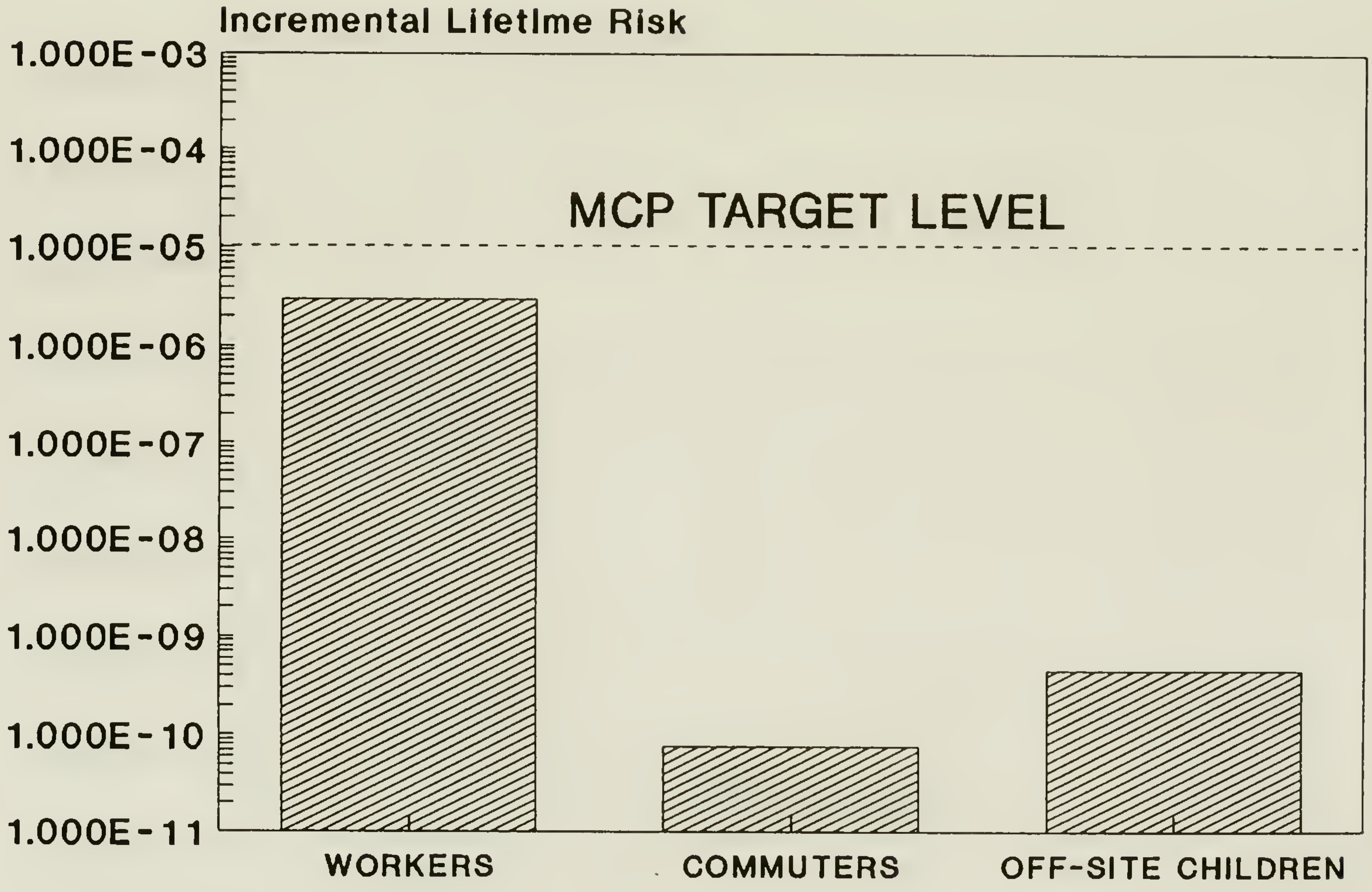


FIGURE ES-2 CARCINOGENIC RISKS
DURING PHASE II CONSTRUCTION

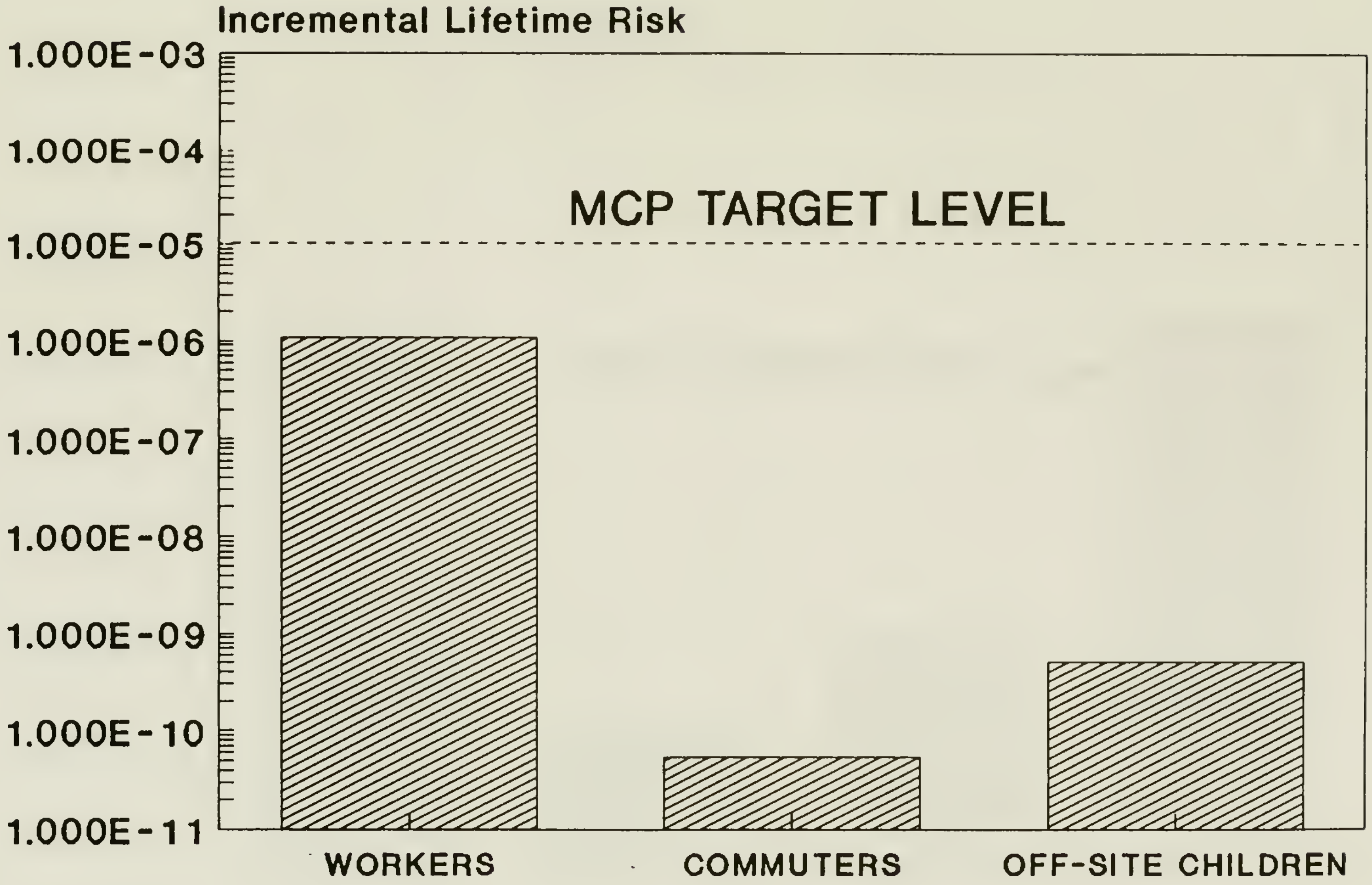
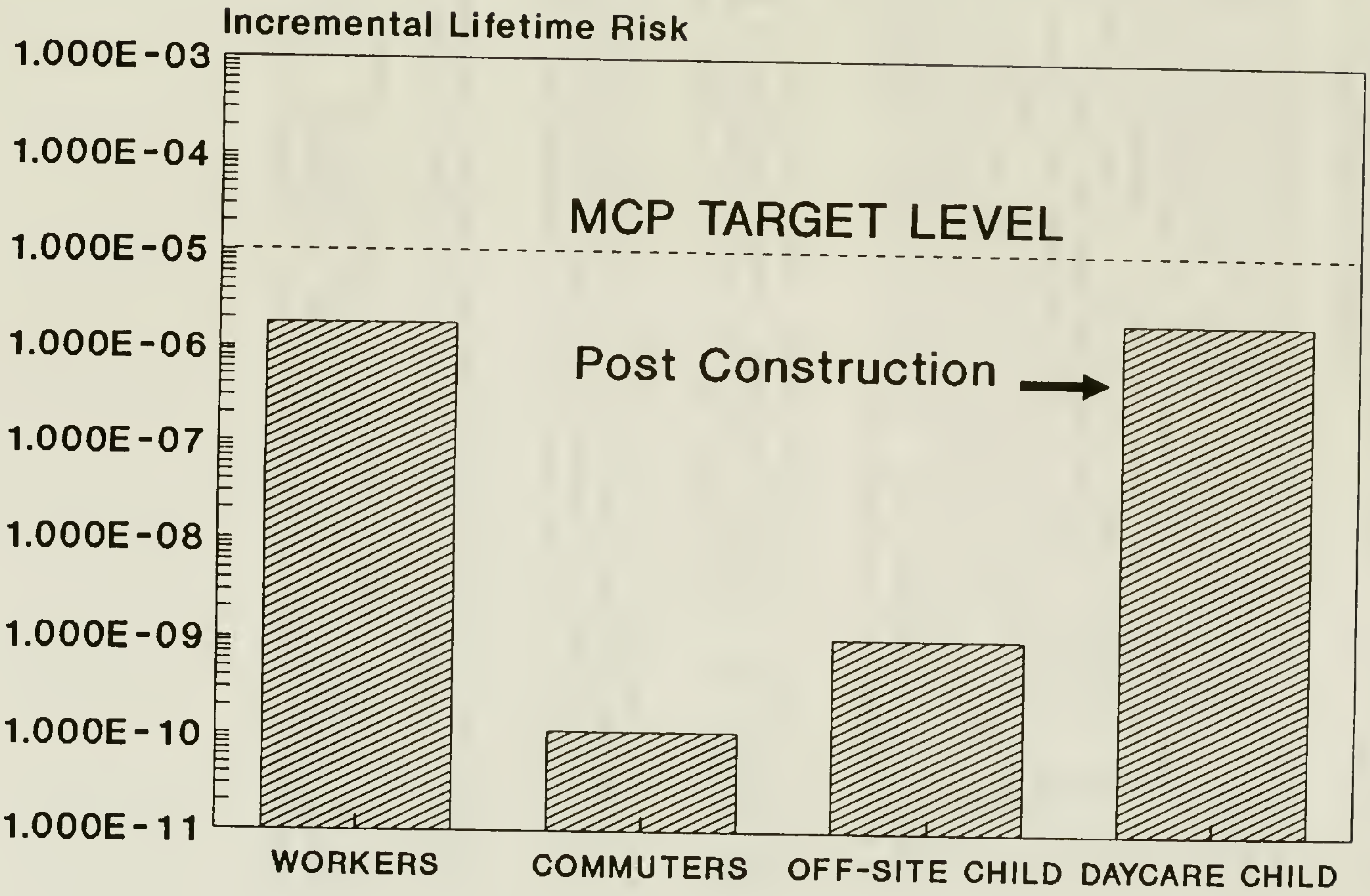


FIGURE ES-3 CARCINOGENIC RISKS
DURING PHASE III



hundred thousand) incremental lifetime risk which is the benchmark used in Massachusetts to judge the "significance" of estimated lifetime risks associated with the presence of chemicals in environmental media.

Risk from non-carcinogens was calculated by dividing the average daily dose of each non-carcinogen by its appropriate RfD and summing the ratios over the entire range of compounds. Figures ES-4 to ES-6 present these risks. All risks are below the 0.2 benchmark level provided by the Massachusetts Contingency Plan (MCP) Guidance Document. Risks to pedestrian commuters and off-site children are one to two orders of magnitude less than the benchmark. Risks to construction workers approach the 0.2 benchmark level. The chemicals driving these risks are thallium and lead in soils. The sum of the hazard index for these two metals is 99.5% of the total hazard index. Lead is in concentrations typical of urban background and is not considered to pose an unusual risk at this site. We have little information on the background levels of thallium. The compound is relatively evenly distributed over the site, and there does not appear to be a "hot-spot".

Risk posed by the site under current conditions or risk to future users such as office workers, emergency workers, or maintenance workers are considered less than the risks due to exposure of construction workers to soils during construction.

Calculated risk to daycare children using the proposed play area in Phase III post construction is also less than DEQE benchmarks. However, we recommend either soil removal or construction of a physical barrier between the play area surface and underlying soils.

The soil gas data indicate extremely low probability of exposure due to intrusion of vapors into above grade structures.

The risk assessment assumes no dust control during construction. Therefore, the estimated hazard index ratio is judged to be conservative. Appropriate dust control measures should ensure that risks do not approach the benchmark value of 0.2 as outlined in the MCP.

FIGURE ES-4 HAZARD INDEX RATIOS
DURING PHASE I CONSTRUCTION

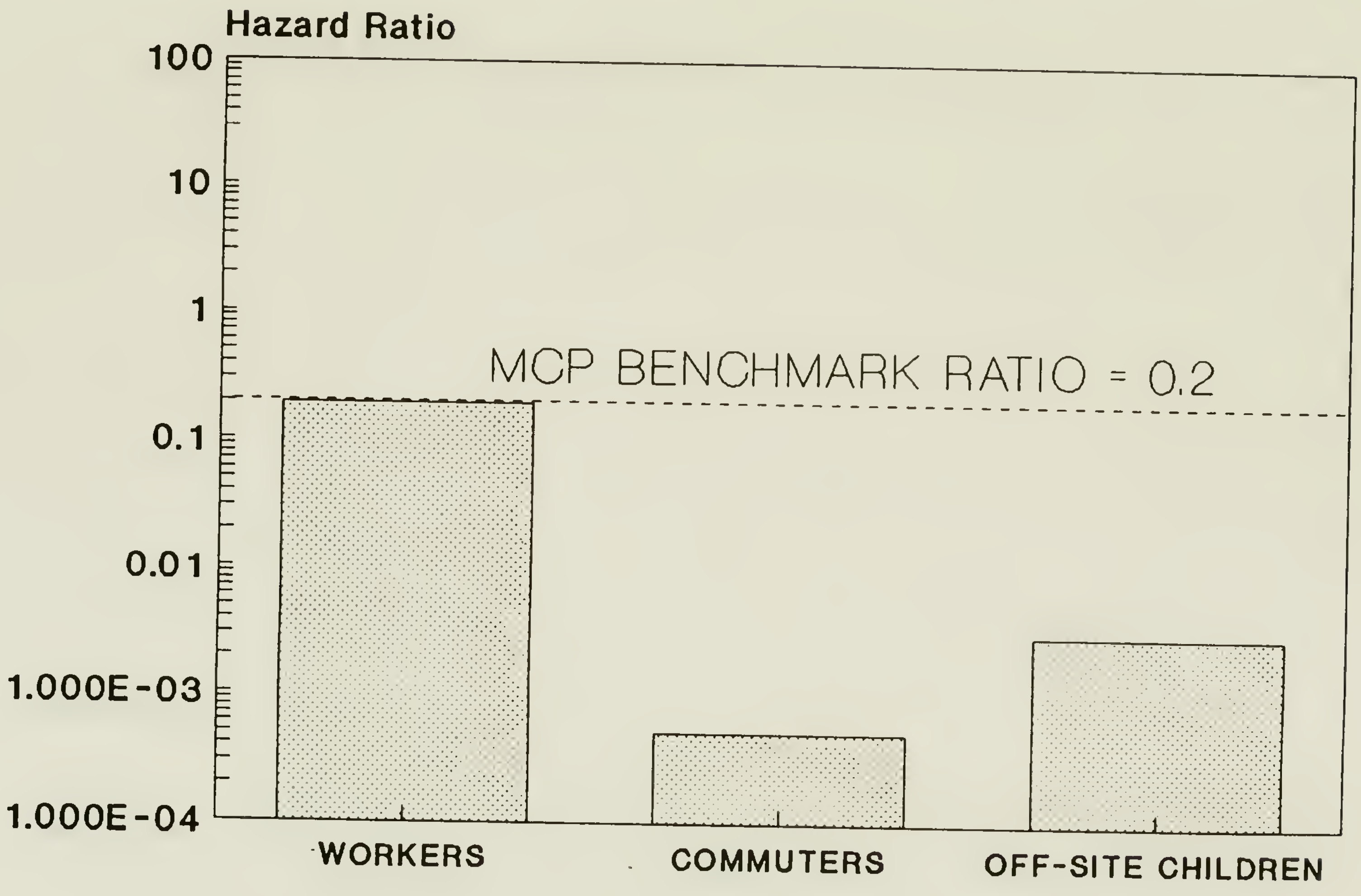


FIGURE ES-5 HAZARD INDEX RATIOS DURING PHASE II CONSTRUCTION

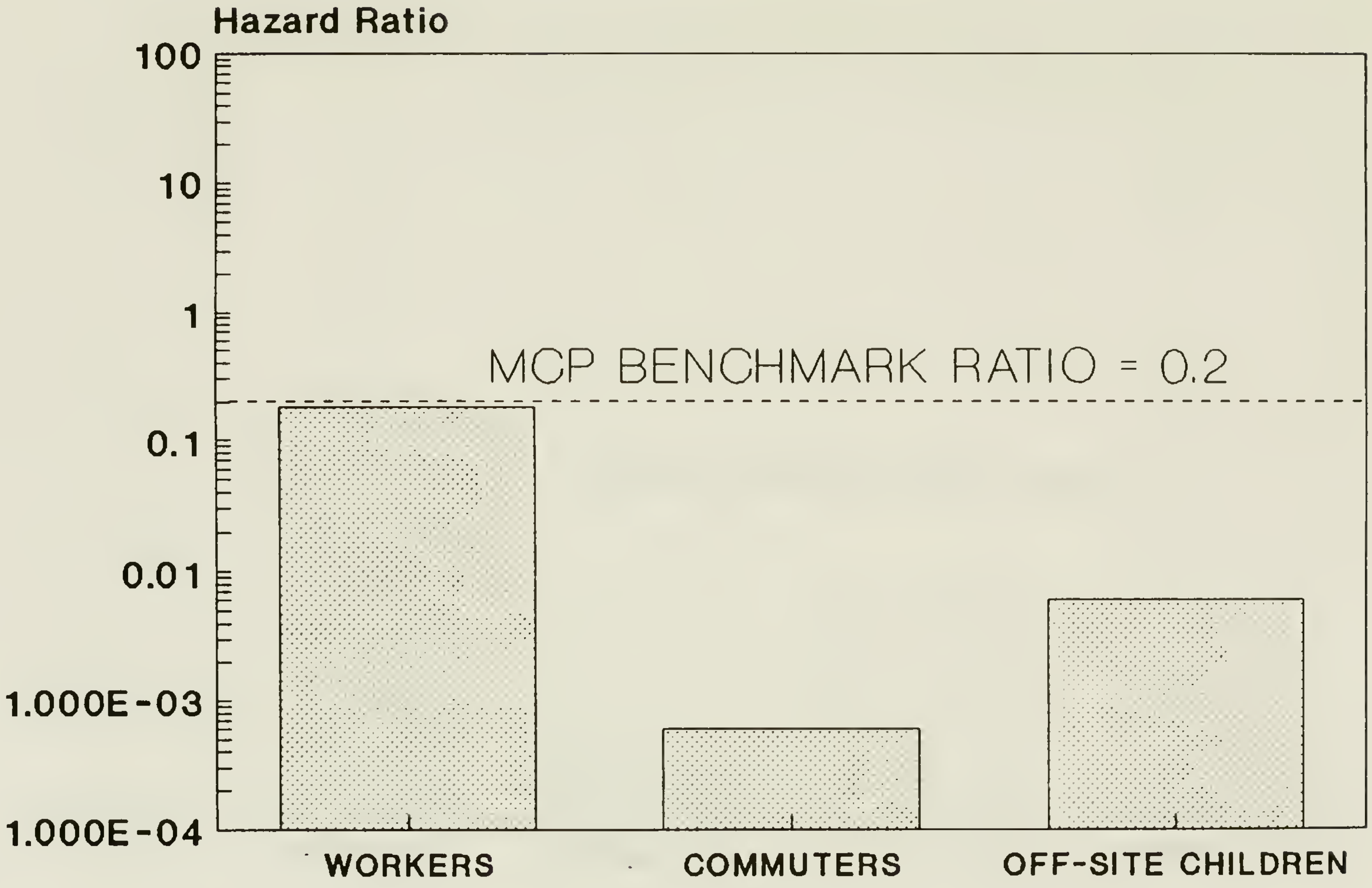
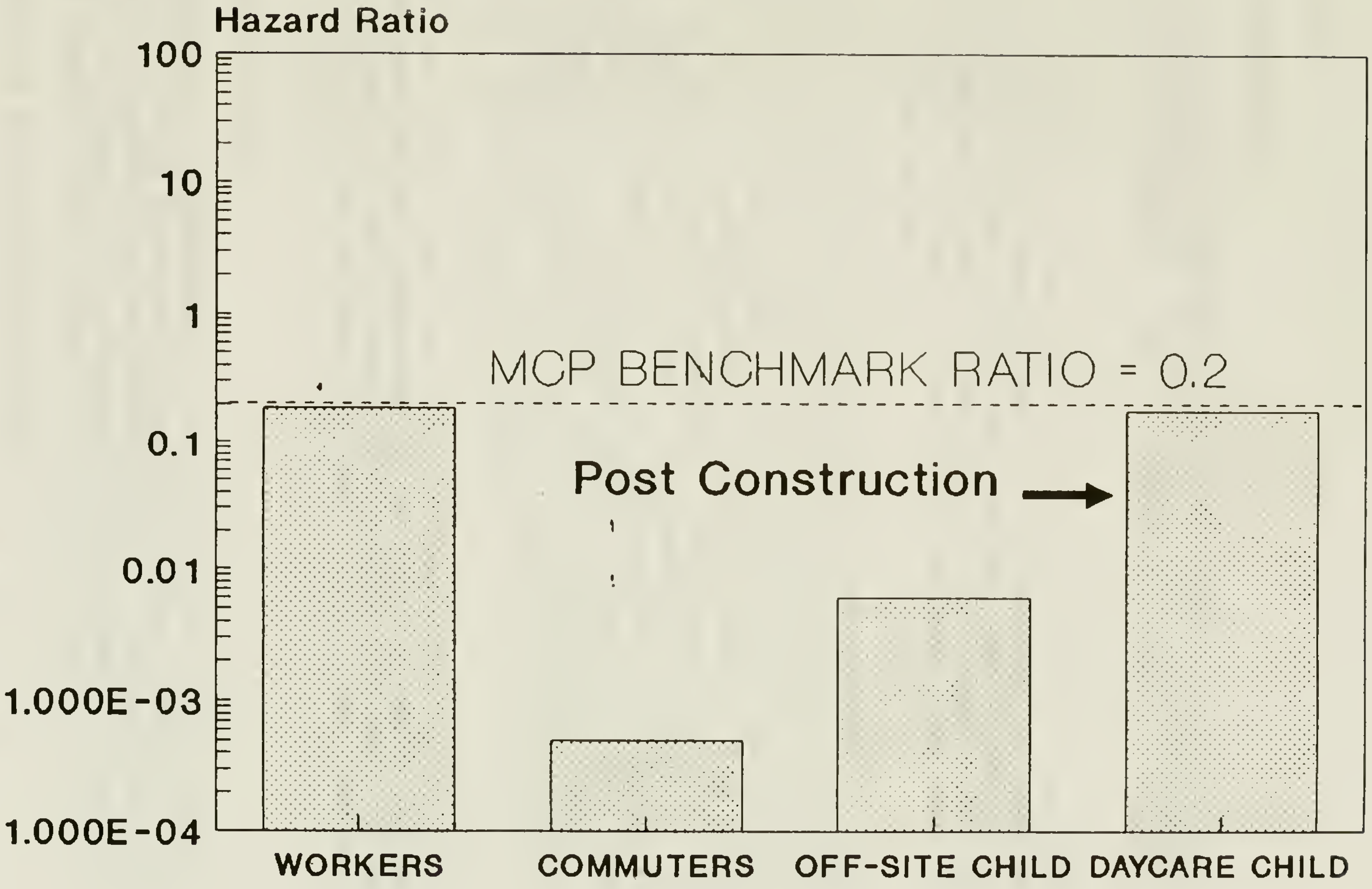


FIGURE ES-6 HAZARD INDEX RATIOS
DURING PHASE III



VOLUME III RISK ASSESSMENT

1. INTRODUCTION

1.1 SITE DESCRIPTION

Volume I of this report describes the site history, surficial and subsurface characteristics at the site. This section briefly describes the surrounding area in terms of potential receptor populations.

The site is adjacent to the Ruggles Street Station of the MBTA Orange Line in Roxbury, Massachusetts. It includes two parcels bounded by Tremont Street to the east, Melnea Cass Boulevard to the north, Ruggles Street to the south, the Ruggles Street Station Building to the west. A pedestrian walkway separates the parcels into three fenced lots.

The immediate surroundings are residential and commercial. The Boston Housing Authority's Community Service Development Project and Services are directly across Tremont Street East of the site. The Whittier Street Development which provides services for the community's youth is approximately one block south of the site. A number of public and educational institutes are in the immediate vicinity of the site. Northeastern University is approximately 1300 feet north. To the northwest is Wentworth Institute (700 ft.). The Harvard Medical School is 1300 feet west and directly south (1300 ft. away) is the Family School. Madison High School and Madison Park are located within 3 blocks southwest of the site. The nearest residential building is approximately 150 feet south of the site.

The pedestrian traffic near the site increases during commuter hours. The area is less frequented by the general public during the day and especially while schools are in summer recess. Those using the walkways have not been observed to enter the fenced-in areas of the site.

1.2 Proposed Site Use

The proposed site development plan includes an office tower, retail stores, and an above ground parking garage. Tower II will have a day care center on the fourth floor. A ground floor day care center is planned for a subsequent office tower. Section 3 provides a summary of the current construction plans.

1.3 Selection of Risk Assessment Method

The Massachusetts Contingency Plan (310 CMR 40.000) and DEQE (1989) describe four methods to conduct a risk assessment.

1.3 Selection of Risk Assessment Method

The Massachusetts Contingency Plan (310 CMR 40.000) and DEQE (1989) describe four methods to conduct a risk assessment. According to 310 CMR 40.545(g), method selection depends upon:

the site physical characteristics;
the contaminant source and extent of release;
the chemical, physical, and biological characteristics of the oil and hazardous materials at the site;
the identification of exposure points;
the determination or estimation of exposure point concentrations;
and,
the results of the Phase II Comprehensive Site Assessment.

The risk assessment must evaluate the risk of harm to public health posed by the site based upon one of the four specified methods. The method chosen must be site specific and not contaminant specific. That is, not more than one method applies to each site. Also, the risk to public health must be based upon current and reasonably foreseeable uses of the site and surrounding environment.

Method 1

Method 1 is a comparison of exposure point concentrations to media specific standards. It applies when there is an applicable or suitably analogous standard for each oil and hazardous material at each current or foreseeable exposure point. The variety of contaminants and the lack of suitable standards for some of these in soil on site precludes the use of method 1.

Method 2

Method 2 requires the use of a specific set of clean-up levels to be promulgated by the State of Massachusetts. At present the state has not published a set of clean-up levels, and therefore method 2 is not applicable to the present site.

Method 3a

Method 3a applies at those sites where oil or hazardous materials are likely to move to exposure points through only one medium. For each contaminant, risk is characterized by reference to applicable or suitably analogous standards, risk based guidelines or policies set by the Department of Environmental Quality Engineering, or by risk based guideline proposed in the risk

assessment. Method 3a is not applicable in the present case because there is more than one media through which contaminants may move.

Method 3b

Method 3b applies in those cases where there may be multimedia transport of contaminants to exposure points and where neither methods 1 nor 2 can apply. Method 3b requires characterizing risk of harm to health by comparing current and reasonably foreseeable exposure point concentrations to applicable or suitable analogous standards for those oil and hazardous materials for which such standards exist. In addition, the risk assessment must calculate the magnitude of current and reasonably foreseeable exposures and estimate the total site cancer and non-cancer risks based upon current and reasonably foreseeable exposure point concentrations and the estimated frequency and duration of exposure to each oil and hazardous material at or from the disposal site. The total site cancer risk limit is one in one hundred thousand over a lifetime and the total site non-cancer risk limit is a Hazard Index of 0.2.

This risk assessment essentially follows method 3b. As indicated in Sections 2 and 3 of this report, available site data provided estimates of the exposure point concentrations, exposure routes, and indicator chemicals. The risk assessment is based upon the risk associated with Chemicals of Interest. These are the most prevalent, mobile, persistent, and toxic chemicals at the site as described in Section 2.2.

2.0 HAZARD IDENTIFICATION

This section identifies the detected compounds on site, compares their concentrations to background, generates a list of compounds used in the risk characterization, and provides the toxicity profiles for those compounds.

2.1 Acute Effects

None of the compounds are expected to be present at levels that would be considered to be acutely toxic (i.e., toxic as a result of a short term exposure on the order of hours or days). This can be demonstrated by considering the potential for construction workers to experience levels of chemicals that may be toxic as a result of acute exposure. In particular, construction workers involved in soil excavation are the individuals most likely to experience any acute effects there may be on this site.

The most likely exposure route leading to potential acute effects is inhalation of dusts or vapors released from excavated materials. Among the chemicals on this site which could produce acute effects if they were present in high enough concentrations are pesticides and volatile organic compounds. The Threshold Limit Values (TLVs) provide a measure of levels below which acute effects are not generally expected in worker populations and are used here as a gauge for assessing the potential for acute effects. The TLV is actually the level that would generally be considered "acceptable" over extended periods of time and thus is often considered "protective" of workers for chronic exposures as well. The TLV values (ACGIH, 1986) for pesticides and volatile organic compounds are given below:

Pesticides	TLV (ug/m3 of air)
Aldrin	250
Dieldrin	250
Heptachlor	500
DDT	1000
Endosulfan	100
Lindane (gamma BHC)	500
Methoxychlor	10000

Volatile Organic Compounds

Benzene	3000	(OSHA Standard)
Chlorobenzene	350000	
Methylene Chloride	360000	
Dichloroethane	810000	
Chloroform	50000	
Toluene	375000	
Ethylbenzene	435000	
Xylene	435000	

These levels can be compared to those calculated for ambient air and soil gas (Section 3 of this report). Such comparisons show that estimated exposure concentrations in air are many orders of magnitude less than the TLVs. Based on this information and experience with these compounds, it appears that the site does not pose any acute hazards and that acute health risks are minimal.

2.2 Identification of Extent of Release of Contaminants

The MCP 310 CMR 40.545(3)(b) requires the risk assessment to describe the horizontal and vertical extent and concentrations of oil and hazardous material at and from the disposal site in all media. Volume I, Section VI of the current report (Haley & Aldrich, 1989) presents the laboratory screening and chemical testing of soils and groundwater, and Volume I Section IV provides the results of the soil gas survey. The media sampled include soil, groundwater, and vadose zone gases. Specific classes of compounds detected include: volatile organic compounds, acid base neutral compounds, metals, and pesticides/PCB's. Sampling of deep soils generally occurred at depths greater than five feet based upon initial headspace screening of soil samples with an HNU photoionization detector and other sample characteristics. Subsequent surface sampling involved a hierarchical random sampling design to characterize the metals, base neutral/acid extractable compounds, total cyanides, and pesticides/PCB's in surface soils.

Groundwater

Table 2-1 summarizes the groundwater data. Among the volatile organic compounds measured in groundwater, only chlorobenzene, acetone, toluene, ethylbenzene, and xylene, occurred above detection limits. The frequency of appearance of these contaminants was low. Ethylbenzene and xylene were present in only four of the fifteen samples taken, while the frequency of occurrence of the remaining volatiles was insignificant at one out of fifteen samples. The volatiles in groundwater were generally in the middle southern portion of Parcel 18-3 (parcel

designations follow Stull and Lee Apportion Plan I, February, 1989-see Figure 2-1). Only one volatile, 3.4 ppb of toluene, occurred in the groundwater in Parcel 18-2. Total volatiles did not exceed 1.12 ppm in any of the individual observation wells at the time of sampling.

Pesticides were generally distributed among the groundwater samples at the site. The most frequently occurring pesticides were alpha-BHC, beta-BHC, and gamma-BHC (lindane). The remaining pesticides occurred in only one or two of eleven groundwater samples analyzed for pesticides. PCB's were not detected in groundwater samples.

Among the metals, nickel or zinc occurred in all groundwater samples analyzed. Concentrations of zinc were within background levels (Hem, 1985), and zinc was well within the Minimum Groundwater Quality Criteria. However the levels of nickel in groundwater were above national averages estimated between 0.3 and 10 ppb (Hem, 1985).

Cyanide, PCB's, and acid and base neutral extractable compounds were not detected in groundwater samples.

Deep Soil

Table 2-2 summarizes contaminant concentration data for the deep soils (soils greater than approximately 4 feet below the surface). Volume I, Section VI (Haley & Aldrich, 1989) provides detailed discussion of the extent of contamination in soils. That section demonstrates that total volatile compounds detected in deep soils range from 2.3 parts per billion (ppb) to 17,700 ppb. The volatile compounds were most numerous and in highest concentrations in soil samples from Parcels 18-3A and 18-3B. Soils from Parcels 18-1A, 18-1B, and 18-2 contained only scattered occurrences of ethylbenzene, xylene, and chlorinated compounds (trichloroethane, trichlorofluoromethane, and methylene chloride).

Pesticides occurred in deep soils throughout the site, and ranged from 12 ppb to 2,080 ppb total pesticides. Detected pesticides included: Alpha-BHC, Beta-BHC, Gamma-BHC, dieldrin, heptachlor, heptachlor epoxide, aldrin, DDT, DDE, DDD, endosulfan, endoslfate, and methoxychlor.

Concentrations of PAH compounds were generally low in subsurface samples (<10ppm). These compounds occurred most frequently on Parcels 18-3A, 18-3B, and 18-2. In the deep soil samples, Parcels 18-1A and 18-1B exhibited few PAH compounds and at low levels (trace to <2ppm total PAH).

Among the metals, arsenic and lead occurred in one boring, B-12 at 7 to 9 feet, at levels above those generally found in North

American soils . Copper, mercury, silver, and zinc occurred at several locations at or above background levels as defined in Figures 2-2 to 2-13. Otherwise, metal concentrations were generally within levels expected in New England soils.

Surface Soils

Volume I, Section VI discusses the distribution of surface soil (soils at depths less than 4 feet) contaminants, and Volume II, Appendices G & H provide the laboratory data. Table 2-3 summarizes the surface soil data. Pesticides were not as common or highly concentrated in the surface samples. Pesticides included: aldrin, DDE, DDT, heptachlor epoxide, and endosulfan. Concentrations ranged from 51 to 428 ppb total pesticides.

The PAH compounds were well distributed over the site. Highest concentrations occurred in soil samples from Parcel 18-1A. This area had the highest total PAH concentrations and the highest concentrations of individual compounds such as pyrene 49 parts per million (ppm), Benzo(a)pyrene (12 ppm), and anthracene (75 ppm).

Metal concentrations are discussed in Section 2.2. Generally, surface soil concentrations were within background (as defined on Figures 2-2 to 2-13) with the exception of mercury at one station.

Surface soil screening of boring samples with a H-Nu photoionization detector generally revealed low levels of total volatiles. In those areas where H-Nu readings exceeded 1 ppm (corrected for background), surface soil gas analysis did not generally reveal the presence of volatile chlorinated compounds or volatiles associated with light petroleum distillates.

2.3 Selection of Compounds of Interest

This section identifies the list of chemical compounds known as the "Compounds of Interest". We will quantitatively evaluate these individually and as a mixture with respect to their health and environmental effects.

The selection procedure for the "Compounds of Interest" identifies the most prevalent, mobile, persistent, and toxic chemicals at the site. Specifically, the selection considers:

- spatial extent, overall quantity, and maximum concentrations in each medium based on monitoring data;
- routes of exposure to contaminants in air, soils and ground water;

- chronic toxicities of the chemicals via these transport routes with special emphasis placed on potential carcinogens. Information from the monitoring data is combined with (i) Risk Reference Dose (RfD) values and Acceptable Intake - Chronic values (AICs) for noncarcinogens and (ii) Cancer Potency Factors (CPFs) for carcinogens to evaluate preliminarily the relative risks posed by the chemicals. In all cases, each chemical that is an EPA or International Agency for Research on Cancer (IARC) Group 1 (known human carcinogen) or Group 2 (probable human carcinogen) is included as a Compound of Interest unless it is a naturally occurring compound present at "typical" ambient concentrations;

- environmental persistence, medium-specific mobility, and ability to bioaccumulate; and,

- exceedances of background, environmental standards or guidance values (used for metals in soils).

Figures 2-2 to 2-16 present site data. Based upon all the above considerations, we selected the following chemical groups for the risk assessment: polynuclear aromatic hydrocarbon (PAH) compounds, pesticides, volatile organic compounds, phthalate esters, and selected inorganic contaminants. All organic compounds identified at the site and many of the metals that exceeded typical background levels are included .

EPA considers the following eight PAH compounds identified at the site as potentially carcinogenic so they are evaluated as a group:

benzo(a)pyrene
benzo(a)anthracene
benzo(b)fluoranthene
benzo(k)fluoranthene
indeno(1,2,3-cd)pyrene
chrysene
dibenzo(a,h)anthracene
benzo(ghi)perylene

Eight other PAH compounds identified at the site are not considered potentially carcinogenic by EPA. We evaluated these with the carcinogenic PAH's for potential systemic health effects based on the two group's common chemical structure. These are:

naphthalene (2-methylnaphthalene was averaged with naphthalene)
fluorene
anthracene
phenanthrene
fluoranthene
pyrene
acenaphthylene
acenaphthene

The PAH compounds are assumed to act additively with regard to potential systemic health effects.

Three phthalate compounds were detected. One of these, bis(2-ethylhexyl)phthalate is considered carcinogenic; the other two di(n-octyl)phthalate and di(n-butyl)phthalate were assessed for potential systemic health effects.

Twelve pesticides and one PCB were detected (at least once) on the site and are included because they could contribute additively to potential effects. These include:

Aldrin
Dieldrin
Heptachlor
Heptachlor Epoxide
DDTR*
Endosulfan I
Endosulfan II
Endosulfan sulfate
Alpha-BHC
Beta-BHC
Gamma-BHC
Methoxychlor

Arochlor 1260
* includes DDT, DDE, DDD

A number of volatile organic compounds were identified in deep soils and/or ground water and are included in the analysis:

Benzene
Chlorobenzene
Acetone
Methylene Chloride
1,2 Dichloroethane
Chloroform
1,1,1 Trichloroethane
1,1,2 Trichloroethane
1,1,2,2 Tetrachloroethane
Toluene
Ethylbenzene
Trichlorofluoromethane
Xylenes

Analyses for a relatively large number of inorganic compounds (in particular metals) were conducted (see Figures 2-2 to 2-13). Many of these (primarily metals) will occur naturally in surface soils. The risk assessor must distinguish between those that are present at elevated concentrations and those that fall within typical background levels for the area or are well within generally accepted soil guidelines. A screening analysis for the inorganic compounds helps focus the assessment on the important organic and inorganic compounds on a site and on the risks that should be addressed as part of the remedial program. The following analysis is used to distinguish between inorganic compounds that should be considered in a quantitative risk assessment and those that can be excluded because they are generally within background levels or well within generally accepted soil guidelines.

Because of the interest in metals in soils, there has already been considerable effort to develop and apply guidelines for "screening" soils based on their metal content. These existing guidelines provide useful tools for evaluating the significance of naturally occurring metals. Based on a review of criteria and typical background values that have been used to screen sites with regard to the levels of chemicals in soils, we have included information in Figures 2-2 to 2-13 that can be used to assess the "significance" of measured levels of metals in soils on Parcel 18. Our sources (referenced in the figures) include standard values for rocks (e.g., shale or granite), values used by New Jersey DEP for assessing elevations above background, and other values considered to be typical of general background ranges. Based on our review of the metals data we have selected the following metals for risk analysis for Parcel 18:

Metal	Rationale for Inclusion
arsenic	one value in deep soils was obviously in excess of typical background levels (Figure 2-2); arsenic may be carcinogenic and therefore the chemical is included; however, it should be noted that the remainder of deep and surface soils are reflective of typical background levels;
copper	a few values for copper in deep soils are at or exceed levels considered typical of the upper range of background (Figure 2-6); however, surface soil values are typical of background conditions;
lead	several values in deep soils exceed levels considered typical of background; one value exceeds 1,000 mg/kg, a level generally considered unacceptable for exposures involving children; however, all surface soils are within general urban background levels as defined on Figure 2-7;
mercury	several values in surface and deep soils exceeded levels considered typical of background (Figure 2-8);
silver	a number of the values for deep soils appeared to exceed typical background levels (Figure 2-11); however, surface soils all appear to be within typical background levels;
thallium	we do not have information on "typical" levels of thallium in soils; however, based on limited information for other sites, the levels of thallium in soils at Parcel 18 was judged to be high enough to include the metal in the analysis; as will be noted later, thallium contributes most of the non-carcinogen risks associated with the site;
zinc	a few of the values in deep soil appeared to exceed levels considered typical of background; however none of the surface soils approached or exceeded these levels.

Soil metals that are not included in the analysis because they are judged to be at typical background levels include:

beryllium (Figure 2-3)

cadmium (Figure 2-4)
chromium (Figure 2-5)
nickel (Figure 2-9)
selenium (Figure 2-10).

Toxicity Profiles

Appendix A provides toxicity profiles for the compounds of interest that are evaluated in this report. The profiles include information on human health and environmental effects, fate and transport data, and guidelines and standards. Several of the profiles also provide information on the levels of the compounds found in various environments.

3.0 EXPOSURE ASSESSMENT

This section develops exposure scenarios by briefly describing the site construction plan and identifying potential human receptors, exposure points, routes of exposure, and exposure point concentrations. We then use these scenarios to estimate average daily dose to each exposed population from each indicator compound.

The quantitative assessment includes four general exposure scenarios: exposure associated with Phase I construction activities; exposure associated with the possible future construction in Phases II and III; and exposure associated with use of the proposed play area.

3.1 Construction Plan

The construction plan for the Parcel 18 and 18B involves three phases. Only Phase I is reasonably well defined. This phase includes construction of the plaza, Tower II on Parcel 18-1B and the right of way parcel, and an above grade parking garage on Parcel 18-2. (Note that parcel designations follow the Apportion Plan 1, Stull and Lee, February, 1989.) The current construction plan anticipates no Phase I construction on Parcel 18-3A or 18-3B, and landscaping or paving on the eastern portion of Parcel 18-1A.

Construction of Foundation And Slab

Three options are being considered for the construction of the foundation for the first office tower. The options differ in the amount of excavation and/or earth moving required. Because contact with soil is a source of potential exposure at Parcel 18 and 18B, such differences among options translate into differences in potential exposure.

Option 1 calls for driving approximately 225 deep piles to bedrock after leveling and grading. Excavation may occur to accommodate the depth of slab. Holes (17" diameter) would be augured to depths of 50-60′ to advance the piles. Soil from the holes will probably be a wet mixture of fill, sand, and clay and could be removed from the site. Additionally, local 4 to 6 foot excavations will occur at each pile cap. It is anticipated that grading may take two weeks and that pile driving work would take approximately 45 days with some additional days early in the schedule for load testing. Overall, we have used a 60 work day period to represent this activity.

During the grading of the site and excavation, there may be 5-10 people on site. During the pile-driving phase, there may be 4

workmen on the ground and 2 men in cranes. Overall we estimate there may be a work force of about 10 to 15 people during the excavation and pile driving stages of the construction.

Option 2 for foundation slab construction is similar to option 1 except that short piles would be installed using "pressure injected footings" (PIF's). There would be more of these piles under option 2 (300-350 PIF's) than deep piles in option 1. Although the number of pile caps would be the same as in option 1, they may be larger.

Option 3 involves densifying the soil via in situ compaction techniques to provide a supportive soil and fill layer for the foundation slab. This option would probably involve much less excavation than options 1 and 2.

Construction of Phase I Tower, Plaza, and Parking Garage

The Phase I construction is an estimated 18 months from early 1990 to mid-1991 under current plans. The above-grade garage on lot 18-2 (Parcel 18B) is expected to take about one year to complete.

It is anticipated that a work force of about fifteen to twenty-five people would be involved in the foundation pile cap construction. The superstructure construction will involve 50 to 100 people. It is assumed that the eastern side of Parcel 18-1A would be used as a staging ground for construction, and, therefore, there would be some disturbance of surface soils in that area (i.e. from truck traffic crew activities).

At the completion of Phase I, it is expected that Tower II, associated retail stores, the plaza, and the above grade parking lot will completely cover the underlying surface soils. It is expected that the eastern portion of Parcel 18-2 would be paved or landscaped and that existing surface soils would be covered.

Phase II/III Construction

Phase II and III construction activities are not well defined at this time. For purposes of this assessment we have assumed that these phases would involve twice as much activity as Phase I and have based exposure estimation on this assumption.

Currently there is no firm time estimate for Phases II through III. In the interim, between Phase I and the initiation of any future phases, the remaining Parcels 18-3A, Parcel 18-3B, and Parcel 18-1A will remain in their present condition which is planted with grasses and small trees and fenced.

3.2 Identification of Potential Human Receptors

This section identifies the potential human receptors regardless of relative magnitude of exposure. The inclusion of a potential receptor here does not mean we included it in the exposure assessment scenarios. If a receptor's potential exposure is obviously much less than that of other receptors or if potential exposure was low due to low or non-detectable exposure point concentrations, we did not perform a quantitative assessment of the less exposed population. We did do quantitative assessments for those receptors which we consider the worst case. For example, we identify maintenance workers and emergency workers as potential future receptors, but recognize that their exposure will be much less than that of a construction worker. Therefore we do not present a quantitative risk assessment for maintenance or emergency workers.

Current Receptors

Under current conditions, the potential human receptors are occasional trespassers on the site. The site is not currently a commercial, industrial or recreational area and therefore does not have regular use.

Potential Future Receptors

Possible human receptors during the construction phases include: construction workers, pedestrian commuters walking the perimeter of the site during construction, and resident children playing off site during construction. We develop scenarios and perform quantitative assessments for these receptors.

After construction, office workers, maintenance workers, and emergency workers (i.e. workers making unscheduled repairs to utilities) are potential receptors. However, as indicated above, the potential exposure is obviously much less than construction workers. Also, we consider that the probability of exposure to an office worker after completed construction is low because the footprint of the buildings and planned landscaping of remaining areas minimizes or eliminates potential exposure to soils. Given the low vadose zone measurements and the planned above grade construction, we do not consider vapor intrusion into completed buildings as a potential exposure pathway. We do not perform quantitative assessments for these populations.

Implementation of later phases does not affect the category of potential human receptors. Phase III includes the construction of a play area for a planned ground floor community day care center in the northwest corner of the site. The children using the playground area are a potential receptor population. We have developed scenarios and performed a quantitative risk assessment for this population.

3.3 Identification of Exposure Points

Potential exposure points include surface soil and subsurface soil.

Although groundwater on site exhibited the presence of contaminants (Table 2-1) there are no plans to use the groundwater on site nor will planned construction activities require dewatering. The Massachusetts Water Resources Authority provides water for the City of Boston, and there are no plans to use site groundwater as a drinking water source. The water table has a depth of about 12 to 22 feet below the surface, and we do not anticipate surface contact after construction. Therefore groundwater is not a potential exposure point.

Although there were several high H-Nu measurements of total volatiles in subsurface and near surface samples from borings, the subsequent soil gas survey (Volume I, Section 4.02) indicate that the measured volatile compounds in the vadose zone were generally below the method detection limits. These limits were in the low parts per billion range. Therefore, we do not consider that the vadose zone is a potential exposure point. In only three measurements of twenty-three were volatiles above the detection limit and these were in the low parts per billion (Table 3-1).

There are no surface water bodies in the immediate area of site.

3.4 Potential Exposure Routes

Present Conditions

Under present site conditions, the only potential exposure route is through incidental contact with site soils by trespassers. There are no exposed subsurface soils or exposed groundwater.

Future Conditions

During Phase I and possible subsequent construction phases, direct contact with soils by construction workers will result in dermal contact and incidental ingestion. Construction workers are generally required to wear long pants and a shirt on site. Dermal contact will be limited to the neck, arms, and hands. The period of maximum dermal contact with soils by construction workers is during auguring for piles, pile cap excavation, and grading. As indicated above, we estimate this period to be 60 days during Phase I.

This sixty day period is also the period of most likely incidental ingestion. Incidental ingestion includes swallowing inhaled dust particles above respirable size as well as soils that may be transferred to the mouth by hand (e.g. wiping the face during work).

Inhalation of respirable particles may occur anytime during construction due to dust-raising site activities. For the purposed of this analysis, We have assumed that no dust control will occur on site.

No dewatering is planned during construction, so groundwater is not considered an exposure point.

Post construction exposure pathways include: contact and incidental ingestion of soils by children in the planned playground area as well as contact of soils by maintenance or emergency workers during future excavation.

3.5 Identification And Estimation of Exposure Point Concentrations

There are two general sampling strata in the soil: the surface soil represented by the composite sampling results in Table 2-3; and the deep soils represented by the discrete sampling results in Table 2-2. To estimate separate exposure point concentrations for each construction phase, we sorted the data based on construction phase location.

Phase I Exposure Point Concentrations

As described above, construction activities during Phase I will involve auguring to advance piles in the area under proposed Tower II, the plaza, and the proposed parking garage (Parcel 18-1B, the Proposed Road Right of Way, and Parcel 18-2) and subsequent staging and storage of equipment in the remaining parcel to the east, Parcel 18-1A (parcel designations refer to Stull and Lee Apportion Plan 1, February, 1989).

During the foundation construction activities, potential exposure points include surface soils, subsurface soils, and airborne particles in Parcel 18-1A, Parcel 18-2, and Proposed Road Right of Way. Table 3-2 provides The exposure point concentrations at these exposure points. The average in the table is the mean of the average deep sample values and the composite surface value. After the foundation is in place, the surface soils on Parcel 18-1A will no longer be an exposure point.

Possible inhalation of dust from the staging area during subsequent Phase I building and garage construction is another exposure pathway. The average surface concentrations in Parcel 18-1A provides exposure point concentrations for on-site and off-site due to inhalation of dust (PM-10) during construction (Table 3-3). We assumed that commuters at the fence line were exposed to the same concentration of dust as on-site construction workers.

The concentrations of compounds in the off-site air were modeled using the near-field box model developed by Pasquill (1975) and Horst (1979). This model is applicable to those exposure scenarios where the receptor group is on site or very close to the site. The box model assumes simple conservation of mass. We have utilized the model to provide an estimate of the height of the mixing compartment with distance down-wind of the site and calculated particle concentration assuming a linear relationship between mixing height and concentration. Vertical mixing is the primary means by which compounds are diluted with distance from the source area over short distances.

The nearfield box model provides a method for determining the height (Hb) of the box at short distances (x) downwind of the site according to the equation:

$$Hb = \ln(0.033 * x^{2.09})$$

The height of the box and associated vertical mixing increases with distance from the source area. For the present analysis, we have assumed that the concentration of fugitive dusts in a 2 m layer of air on the site is 90 ug/m3. This number is based on data obtained by the Transportation Research Board (1978) which provided data as total suspended particulates (TSP). Available monitoring data for New England (USEPA, 1989) indicates that PM10 (particles less than 10 um in diameter) comprise 40% of the TSP. To be conservative, we used 50% of the TSP to derive the value of 90 ug/m3. Based on a review of available data for Massachusetts and other New England states, we believe this number will likely overestimate actual PM10 levels associated with the construction activity and therefore is viewed as a conservative estimate. We then estimated the degree of dilution of this material with distance. For example, at a distance of 100 m, the height of the box is now calculated to be 6.2 m. This represents a "dilution factor" of 6.2/2 or 3.1. Thus, we estimate that the level of PM10 associated with construction is 90 ug/m3 divided by 3.1 or about 30 ug/m3 at a distance of 100 m. This is the approach used to estimate exposure levels of fugitive dusts at off-site receptors.

Phase II And III Exposure Point Concentrations

Similarly, the average of surface and subsurface concentrations in the Parcel 18-1A soils provide exposure point concentration for risk assessment associated with foundation construction during Phase II (Table 3-4). Phase III exposure point concentrations during foundation construction are the averages of surface and subsurface contaminants from soils on Parcel 18-3A, Parcel 18-3B, and the Playground Easement (Table 3-6). To assess risk due to inhalation during subsequent building construction for the proposed Phases II and III, we used the average of surface soil contaminant concentration in Parcel 18-3B and 18-3A (Table 3-5).

We calculated off-site fugitive dust concentrations for an off-site residences approximately 50 meters from the Phase II and Phase III work areas using the model described above. Modelled off site fugitive dust concentrations at this distance were 40 ug/m3.

3.6 Development of Exposure Scenarios

Current Exposure Scenarios

Potential current exposure scenarios include the incidental ingestion or inhalation of surface soils by occasional trespassers. The site is currently fenced and landscaped. Based on discussions with individuals familiar with local activities (Boston Housing Authority Community Service Development), it appears that the site is not currently used by local residents for recreational or other purposes. There are recreational facilities available in the immediate area. Therefore, such exposure would be infrequent relative to the level potentially experienced by workers during construction when site soils will be disturbed and activity will be regular and extensive. We therefore did not quantitatively assess risk under current conditions since it will be considerably less than the potential risk to on-site and off-site receptors during the construction period. The risk to trespassers is conservatively included within the range of risks calculated for construction workers and pedestrian commuters as described in succeeding sections.

Future Exposure Scenarios

Future exposure scenarios include: exposure to construction workers and off-site receptors during various construction phases; exposure to children attending the proposed Phase III day care center from the soils in the proposed playground; exposure to maintenance and emergency workers during utility excavations; and exposure to office workers occupying the proposed buildings. Exposure of officer workers in the future to site soils is unlikely because proposed site development plans involve covering most of the site with either asphalt, concrete, or landscaping (topsoil and plantings). Therefore office workers would be physically excluded from contact with surface and subsurface soils present at the site. Further, any exposure to office workers would be less than construction worker exposure.

The soil gas data indicate that the vadose zone is an unlikely exposure route for office workers occupying the site. The present plans involve construction on a concrete slab. There will be no subsurface rooms that could potentially collect gases present in the surrounding soil. Further, the levels of soil gases in the vadose zone were observed to be generally non-detectable or very low and are not considered to represent a strong source for buildings that are constructed above grade. Therefore, the

exposure of office workers to entrained soil gases was judged to be insignificant at this site. We therefore did not develop a quantitative assessment of office worker exposure either from soils or soil gas exposure.

Similarly, the potential exposure of maintenance and emergency workers to site soils in the event of excavation of utility lines or other emergency procedures which require soil disturbance will result in less exposure and less frequent exposure than that of construction workers. The risk to such workers will be less than that calculated for construction workers. We did not therefore develop a quantitative assessment of maintenance or office worker exposure to excavated soils.

The scenarios for which we made quantitative assessments include:

Scenario I - Phase I Construction Worker Exposure

> Phase I Foundation Construction - soil ingestion, direct contact and inhalation from excavation soils
>
> Phase I Building Construction-soil ingestion, and inhalation from staging area soils

Scenario II Phase I Commuter Exposure

> Phase I Foundation and Building Construction - inhalation of airborne soils by pedestrian commuters

Scenario III Phase I Off Site Children Exposure

> Phase I Foundation and Building Construction - inhalation of airborne soils by children at off-site playground

Scenario IV - Phase II Construction Worker Exposure

> Phase II Foundation Construction - soil ingestion and direct contact
>
> Phase II Foundation and Building Construction - inhalation of airborne soils from staging area (Note that in Phases II and III, we assumed that inhaled soils were from staging areas since surface concentrations in these areas were slightly higher than in the area of piling excavations.)
>
> Phase II Building Construction-soil ingestion from staging area soils

Scenario V - Phase II Commuter Exposure

Phase II Building Construction - inhalation of airborne soils by pedestrian commuters

Scenario VI - Phase II Off Site Children Exposure

Phase II Building Construction - inhalation of airborne soils by children at off-site playground

Scenario VII - Phase III Construction Worker Exposure

Phase III Foundation Excavations - soil ingestion and direct contact

Phase III Building Construction-soil ingestion from staging area soils

Phase III Building Construction - inhalation of airborne soils by construction workers

Scenario VIII - Phase III Commuter Exposure

Phase III Building Construction - inhalation of airborne soils by pedestrian commuters

Scenario IX - Phase III Off Site Children Exposure

Phase III Building Construction - inhalation of airborne soils by children at off-site residence.

Scenario X - Children Using The Proposed Phase III Playground

Each of these scenarios has various assumptions and parameters to calculate average daily dose. Tables 3-7 to 3-22 summarize these.

3.7 Estimation of Average Daily Dose

Average Daily Dose (ADD) is the total intake of contaminant by the exposed individual normalized to body weight and appropriate "averaging period". For carcinogenic risk, the averaging period is a lifetime, seventy years. For systemic health risk, the averaging period is taken to be the period of exposure, and it changes with scenario as indicated in Tables 3-7 to 3-21. Since we are looking at chronic health effects, this is an appropriate and probably conservative approach. The reference dose values (RfD) themselves are based on life time exposures.

For soil ingestion, the average daily dose is:

$$ADD = \frac{C * I * AF * DUR}{BW * AP}$$

where,

ADD= Average Daily Dose
C= Concentration of Contaminant
I= Ingestion Rate
DUR= Exposure Duration
BW= Average Body Weight
AP= Averaging Period.
AF= Absorption Factor

For dermal contact with soils average daily dose is:

$$ADD = \frac{C * SA * MS * AF * DUR}{BW * AP}$$

where,

SA= exposed skin area
MS= mass of soil per unit skin area.

For inhalation, the average daily dose is:

$$ADD = \frac{C * VR * AF * DUR}{BW * AP}$$

where,

C= Concentration of Respirable Soil Particles in Air (mass/volume) Multiplied by the Contaminant Concentration (mass/mass)
VR= respiratory volume.

We summed the average daily doses for soil ingestion and dermal contact for the risk characterization, and treated the inhalation average daily dose separately because it often has a distinct reference dose (RfD). Tables B-1 to B-13 (Appendix B) provide the average daily doses for each exposure pathway and receptor. Note that Table B-14 provides all the coefficients used in the calculations.

In Phase I and II, the averaging period was the 18 months of construction for all scenarios. We assumed Phase I inhalation exposures to workers from excavated soils occurred for 60 days, and the inhalation exposure from the staging soils area was 300 days because soil concentrations in the area of foundation construction were higher than staging area soils for this phase. This provides a total of 360 days of exposure over a 540 day averaging period. We assumed that the Phase II inhalation exposure was entirely from the staging area for 360 days over a

540 day period since in that case the staging area surface soil concentrations were higher than the surface soil concentrations in the area of building construction. This is conservative since the staging area soils have a higher average concentration for most contaminants than the Phase II building site soils. In Phase III, we doubled the exposure and averaging period because this proposed phase will entail constructing two towers.

In all phases, we assumed that pedestrian commuters would spend twenty minutes each day exposed to on-site air, and that off-site children would play for 4 hours each day in the nearby playground or just outside the nearby residences during the entire period of construction.

For daycare children using the proposed on-site play area, we assumed that an individual child would use the area for seven months of the year for a five year period.

4.0 DOSE RESPONSE ASSESSMENT

4.1 Overview

This section of the document provides information on the health and environmental effects of site-specific Compounds-of-Interest. This information provides a basis for dose-response assessment. In combination with information developed in the exposure assessment it allows estimate of risks.

Evaluation of a chemical's potential for toxicity involves the examination of available data that relate its observed toxic effects to doses at which they occur. Generally, there are five categories of information that are considered in this part of a quantitative risk assessment:

1. Information on the potential for chemicals to initiate or promote cancers.
2. Information on the potential acute or chronic non-cancer effects of chemicals.
3. Information on the environmental effects of chemicals.
4. Information on potential aesthetic effects.
5. Information on applicable air, surface water, and groundwater standards.

Appendix A which includes Chemical Profiles provides much of this information.

A wide variety of factors must be considered in using data in a quantitative toxicity assessment. As will be discussed in following sub-sections, there may be a variety of relationships between dose and effect. However, an important concept that should be considered is that some chemicals display thresholds (i.e., there are doses below which the chemical does not cause an effect). In general, non-carcinogenic (acute or chronic systemic) effects are considered to have threshold values while carcinogenic effects are considered not to have thresholds. Toxicity studies for the former tend to focus on identifying where this threshold occurs. Studies for the latter tend to focus on identifying the slope of the linear portion of dose-response curve at low doses. In accordance with current scientific policy concerning carcinogens, it is assumed/presumed that these curves pass through "0" indicating that any amount of dose has some associated response. This is what is meant by a non-threshold effect. Scientists tend to agree that this is a sound policy for many compounds; for other compounds (e.g., some of the chlorinated solvents) there is debate concerning the no-threshold concept for

carcinogens. In this assessment, the no-threshold approach is taken for all probable carcinogens.

A detailed discussion of toxicological principles associated with dose/response assessment is not provided here. However, the following references were relied upon in providing background discussion of some of the basic information:

> U.S. Environmental Protection Agency. 1987. Integrated Risk Information System Supportive Documentation. EPA Report No. 600/8-86/032a.
>
> ICAIR, Life Systems. 1985. Toxicology Handbook: Principals Related to Hazardous Waste Site Investigations. Submitted to Office of Waste Programs Enforcement, U.S. Environmental Protection Agency, Washington D.C.

4.2 Chemical Profiles

Appendix A, Chemical Profiles, summarizes information on the basic physical, chemical, and toxicological data used in assessing health and environmental effects. These profiles provide more detail on the chemicals and serve as resource documents. Further, the appendices include information on target organs for specific effects.

Primary sources of data for health effects of Compounds of Interest include:

- the EPA's Integrated Risk Information System (IRIS) Data Base; a computer search of the data base was made for the Compounds of Interest as part of this project;
- the EPA's Superfund Public Health Evaluation Manual;
- Agency for Toxic Substances and Disease Registry (ATSDR) profiles for selected compounds;
- personal communications with the EPA's Cancer Assessment Group (CAG);
- numerous reports prepared in support of drinking water criteria, OSHA standards, and water quality criteria as well as comprehensive reviews of the literature for specific compounds.

4.3 Assessment for Potential Carcinogens

This sub-section provides information that can be used to characterize the carcinogenic potencies of Compounds-of-Interest at Parcel 18. Values for cancer potencies presented in this sub-section, when combined with data on exposure, are used to make quantitative estimates of risk.

The EPA's Cancer Assessment Group (CAG) in Washington D.C. is the primary source of information on the cancer potencies of selected compounds. As a matter of EPA policy, estimates of the carcinogenic potency of chemicals are based on the assumption that there are no threshold levels and that the response is linear with dose at low levels (at those encountered in the environment). Thus, there is always some level of risk at every exposure concentration. For this reason, many regulatory actions at sites and risk assessments tend to focus on the potential carcinogens. Under most circumstances, these chemicals tend to "drive" the risk assessment, because the chemicals do not have thresholds. The "weight of evidence" regarding the potential carcinogenicity of a chemical varies as a result of variations in the available test data, adequacy of studies, kinds of studies, and observed responses. The EPA has established weight-of-evidence categories for characterizing chemicals (Table 4-1). In the subsequent discussion, the weight of evidence will be presented for selected Compounds-of-Interest.

Compounds-of-Interest that are evaluated with regard to potential carcinogenic effects are presented in Table 4-2. Many of the chemicals are PAH compounds. In addition, pesticides, benzene, bis(2-ethyhexyl)phthalate, and a few chlorinated solvents may have carcinogenic effects.

The carcinogenic potency of a chemical can be expressed in a variety of ways. In this document, the potency is expressed as a "potency factor". The potency factor as calculated by the EPA CAG is usually the 95% statistical upper bound on the slope of the dose-response curve in the low-dose linear portion as estimated by the multistage linearized model. The response is the lifetime risk of cancer in humans as a function of an average daily exposure level (mg/kg/day) occurring throughout that lifetime. The slope or potency factor is expressed in units of $(mg/kg/day)^{-1}$. Cancer potency factors for some of the Compounds-of-Interest have been estimated by EPA's Cancer Assessment Group (CAG), are available in EPA's Health Effects Assessment documents, and, if fully accepted by EPA are included in the IRIS data base.

Many of the potential carcinogens at the site are PAHs. At present, the EPA has published a potency factor for only one of these, benzo(a)pyrene (B[a]P). In estimating the risks associated with other carcinogenic PAH compounds, B(a)P is often used as a surrogate for the other compounds. It is well recognized that this

will overestimate the risks because B(a)P is one of the most potent chemicals among carcinogenic PAHs. Several groups have been working toward developing relative potency values for other PAH compounds. Chu and Chen (1984) with EPA's Cancer Assessment Group (CAG) have carried out an evaluation and estimation of potential carcinogenic risks of PAHs. They point out the limitations of existing and proposed approaches.

Most recently ICF Clement Associates (1987) has conducted an analysis and prepared a report on the potencies of various PAH compounds relative to B(a)P. ICF Clement Associates (1987) also present a biological basis for the carcinogenic processes and develop a predictive model based on this underlying mechanism. Using a biologically-based model, they derive a new CPF for B(a)P. The model and new CPF are still under review and, based on discussions with EPA's CAG and several regional offices where the methods have been considered, we do not propose to use the new CPF in the present assessment. The CPFs that had initially been developed by EPA will be used. We note that these CPFs are not included within the EPA's IRIS data base because of the re-evaluation of data for B(a)P. However, we will be using the information that has been gathered by and for EPA on the relative potencies of the various PAH compounds.

The relative potencies for other PAH compounds were derived by ICF Clement based on an evaluation of data from nine experimental studies. Estimates based on the most reliable of these studies are summarized in Table 4-3.

A summary of the cancer potency factors that are used in the quantitative risk assessment is given in Table 4-4. The values provided in this table are used in the equations for estimating risk provided in Section 5.

Potency factors for PAH compounds are provided for EPA's estimate for B(a)P, and for other PAH compounds based on their potency relative to B(a)P. In addition, potency estimates derived by the EPA CAG for the other compounds are also provided. Most of the information is taken from a June, 1989 search of the EPA IRIS data base. Additional information was obtained from the ATSDR and EPA drinking water documents.

4.4 Assessment for Non-Carcinogens

All the Compounds-of-Interest have the potential for effects that are non-carcinogenic in nature. Therefore, when evaluating such effects potential carcinogens should be included along with chemicals that are typically considered to be non-carcinogens.

The assessment of non-carcinogenic effects is complex. There is a broad interaction of time scales (acute, subchronic, and chronic)

with kinds of effects. In addition, there are various levels of "severity" of effect.
In this risk assessment we will focus on evaluating the consequences of subchronic and chronic (long term) exposure to various chemicals. None of the chemicals are expected to be present at levels that would be toxic as a result of acute (short-term) exposure.

This sub-section provides information that is used to assess the potential subchronic and chronic health effects associated with the presence of Compounds-of-Interest at Parcel 18. Information presented here is used to estimate risks using the equations and methods outlined in Section 5 of this document.
The potential subchronic and chronic effects of the Compounds-of-Interest are described in the chemical profiles included in Appendix A. The subchronic and chronic effects of Chemicals-of-Interest can be evaluated by reference to:

- Acceptable Intake Subchronic (AIS) values for exposures on the order of weeks to months, and,

- Acceptable Intake Chronic (AIC), or Risk Reference Dose (RfD) values for chronic exposures on the order of a year to years.

In the analysis presented in this report, we have generally used the RfD or AIC values to assess subchronic as well as chronic health risks.

These values serve as benchmarks for assessing the potential for non-carcinogenic health effects. They represent "threshold" health effects values below which no effects are expected. To ensure that these benchmarks are set low enough, uncertainty in the supporting data base is taken into account through the application of uncertainty or safety factors.

The health-based criteria used in this report are generally reference doses published in the EPA's Integrated Risk Information System (IRIS) database or Acceptable Intake Chronic (AICs) published in the EPA Superfund Public Health Evaluation Manual. A reference dose is defined in IRIS as an estimate (uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. A critical effect refers to the health endpoint upon which the reference dose is based. The uncertainty factor contributes as a divisor to the dose associated with the critical effect, which is usually a No-Observed-Adverse-Effect-Level (NOAEL) or a Lowest-Observed-Adverse-Effect Level (LOAEL). Most uncertainty factors are standardized and include:

- 10-fold factor for extrapolation from animals to humans;

- 10-fold factor for variability in the human population;
- 10-fold factor for use of a less-than-chronic study;
- 1 to 10-fold factor for extrapolation from a LOAEL.

The use of ten fold uncertainty factors is traditional. However, there may be situations where data support the application of smaller uncertainty factors. There is on-going research directed at the use of physiologically based pharmacokinetic modeling for interspecies extrapolation. However, at this time, no specific guidance can be provided on the use of this method for developing better extrapolation (from animal to human) values for application to the identified Compounds-of-Interest.

Modifying factors also contribute as divisors to the NOAEL or LOAEL and are usually one; however, in certain instances, the IRIS review group uses, based on collective professional judgment, a modifying factor to further adjust the reference dose. Confidence in the reference dose refers to a qualitative judgment of the confidence that the EPA review group had in the quality of the critical study, the supporting database, and the reference dose. A "low" designation suggests that the reference dose is likely to change as new information becomes available.

The result of applying various multiples of 10 is that for many chemicals the AIC is calculated to be a factor of 100 less than the NOAEL and for some (e.g., ethylbenzene) as much as a factor of 1000 less.

EPA has published no AIC or RfD values for PAH compounds. However, these are among the most common compounds found in soils on Parcel 18 and 18B. Using the same methodology and approach used to derive RfDs, Environ (1986) estimated AIC values for selected PAH compounds. A summary of these values and the underlying bases for the numbers are presented in Table 4-5. As can be seen from the table, very large uncertainty factors (1,000 to 10,000) have been used to estimate AIC values from limited studies on animals. The values range from 0.0053 mg/kg/day to 0.2 mg/kg/day. An average AIC value for these PAH compounds is 0.07 mg/kg/day. In this analysis, we have selected 0.01 mg/kg/day as an AIC value for PAH compounds. This is less than most of the AIC values derived by Environ (1986). Naphthalene was calculated to be less than 0.01 and we use the calculated value of 0.005 mg/kg/day for this compound. However, a note of caution is offered on the use of the Shopp et al. (1984) data for naphthalene. The authors stated that the CD1 mouse, the test animal in their study, is not a good animal model for cataracts or hemolytic anemia, the two adverse effects of naphthalene observed in humans. This creates problems in species extrapolation.

Because of the margin of safety built into the AIC value, exceedence of the number has no immediate meaning with regard to specific health effects, the frequency of effects, or the magnitude of effects. However, exceedence of the number should serve as an indicator that the potential for unacceptable exposure does exist and precautions should be taken to limit exposure. The Massachusetts MCP guidance calls for a comparison to a benchmark of 20% of the RfD or AIC value. This approach takes into account the possibility that individuals may be exposed to these same chemicals from other sources. The basic assumption here is that individuals may experience 80% of the AIC or RfD exposure as part of everyday living (all other sources), and, therefore the incremental increase in exposure should be limited to 20% of the RfD or AIC. This is not always a good assumption and can be evaluated directly. However, in this assessment, this approach will be used unless otherwise specified.

RfD or AIC values are generally not available for all exposure routes (oral, dermal, inhalation). If a value is available for only one route of exposure we have used it for the other routes unless there is a strong reason to do otherwise. This is a source of uncertainty in the analysis but at least ensures that some account is taken of the presence of chemicals in various exposure routes.

The RfD or AIC values used in this analysis are presented in Table 4-6.

4.5 Environmental Effects

Information on environmental effects of the Compounds of Interest is provided in Appendix A. However, the site is not near any natural habitats (open space, wetlands, forests, surface water bodies) and groundwater does not surface at wetlands or surface water bodies near the site. Post construction activities will not change the potential for environmental impact or groundwater flow direction and, therefore, "ecological risks" are not evaluated with regard to this particular site.

5.0 RISK CHARACTERIZATION

5.1 Comparisons of Measured or Estimated Concentrations to Applicable Guidelines and Standards

Ground Water

Ground water sampling results are described in Volume I "Report on Phase Two Oil and Hazardous Material Site Evaluation Parcel 18 and Parcel 18B Boston Massachusetts" (Haley & Aldrich 1989). The report noted evidence of some hydrocarbon contamination in selected wells. Petroleum sheen or odors were observed at seven of twenty-five observation well locations. Phase-separated petroleum product was observed in sampling well B7-OW. As noted in Volume I, "the product in Well B7-OW was observed to be amber colored and may be related to a petroleum product identified as kerosene in test pit TP-19, approximately 30 feet to the west".

Groundwater was sampled from fifteen of the twenty-one observation wells during February 1989. Results are compared to applicable guidelines or standards in Table 5-1. With few exceptions, levels of chemicals were within applicable guidelines or standards.

PAH Compounds

PAH compounds were not detected in groundwater.

Pesticides

Low levels of eight pesticides were detected. Heptachlor exceeded its MCL of 0.4 ug/l in one sample where it was detected at 0.5 ug/l. The compound was detected in three of twelve ground water samples.

The BHC compounds (including Lindane - gamma-BHC) were observed in all the ground water samples. EPA has published an MCL for lindane of 0.2 ug/l. Levels of lindane (gamma-BHC) were at or above this level in two samples. Levels of other BHC compounds for which there exists no MCL also exceeded this level (0.2 ug/l) in ten of the twelve samples. The highest concentration of BHC compounds was observed in sample B7-OW at 5 ug/l.

Volatile Organic Compounds

Low levels of VOCs were observed in ground water. None of these levels exceeded applicable guidelines or standards.

Metals

With one exception, none of the metals in ground water exceeded any of the applicable guidelines or standards. Nickel has a Maximum Contaminant Level Goal (MCLG) of 100 ug/l. This level was exceeded in all five of the samples analyzed from the site. The maximum concentration observed was 230 ug/l.

Air

Concentrations of chemicals in air at the site were estimated during construction periods by multiplying the average surface soil concentrations of PAH, pesticide, and metal compounds by an estimated suspended dust level of 90 ug/m^3. These estimates were developed for each phase of construction. The highest of the average values for each phase are presented in Table 5-2 along with the Massachusetts Allowable Ambient Level (AAL) and the Allowable Threshold Concentration (ATC). All units are in ug/m^3. Massachusetts has developed an AAL for only one of the PAH compounds, naphthalene. The estimated concentration of this compound in site air during construction is many orders of magnitude less than the AAL. Massachusetts has estimated an AAL for two of the pesticides - heptachlor and lindane (gamma-BHC) - and neither is estimated to exceed the AAL or ATC value in ambient air.

Among the metals, lead is the only compound which appears to have a potential for exceeding its AAL of 0.07 ug/m^3. The estimated concentration of lead in the air is 0.1 ug/m^3 which is a little above the AAL but less than the ATC of 1 ug/m^3. The estimated concentration of 0.1 ug/m^3 would be less than (within) the primary public health standard of 1.5 ug/m^3. The ambient level of lead in air within Boston is about 0.1 ug/m^3 (USEPA, 1988).

Measurements of VOCs have been made in surface soils via soil gas analysis by Gas Chromatograph. Twenty three samples were collected and analyzed from areas where elevated levels of volatiles had been indicated by HNU measurements. Table 5-2 presents the maximum values detected in the soil gas survey. Measured soil gas levels were generally below the limits of detection and would be considered to be at levels that would not act as strong "sources" to the overlying air mass or to indoor air levels within the buildings constructed on Parcel 18. We note, however, that levels of 1-1-1 trichloroethane (suspected to be present) and toluene could exceed their AAL values within the soil gas but are less than (within) the ATC values. Concentrations of these chemicals would be reduced in concentration upon emission to the overlying air mass and would not be detectable.

Elevated levels of volatiles were detected with the HNU in deeper

soils on the site (Haley and Aldrich, 1989). If large and deep excavations were being planned for the Parcel 18 site there would be a greater potential for release of VOCs. However, with the exception of driving piles and placement of the foundation slab, no deep excavation or construction is planned.

5.2 Estimates of Human Health Risks

Estimates of risks were developed for individual carcinogens and non-carcinogens as well as for mixtures. The latter is necessary because a variety of chemicals are found at the Parcel 18 and 18B Site. The quantification of potential risks is based on the guidance published by the EPA (50 FR 1170 et seq., 9 January 1985) and by the Commonwealth of Massachusetts in "Guidance for Disposal Site Risk Characterization and Related Phase II Activities" (DEQE, 1989). Risk estimates for chemical mixtures are generally based on the assumption of additivity unless there is information to the contrary.

In accordance with current guidance, the assumption of additive effects is generally made for non-carcinogens in order to calculate a Hazard Index Ratio. However, it is also generally recognized that this may not be a good assumption because of differences in target organs, application of uncertainty factors, and differences in modes of actions of chemicals. However, the Hazard Index is currently identified in existing guidance as a means of identifying situations where additional attention should be given or remedial actions taken.

All estimates of risk have been calculated using computer-based spreadsheets. The calculations are presented in Appendix B. Note that for the reader's convenience, Table B-14 lists all the coefficients used in the calculations.

5.2.1 Estimation of Carcinogenic (Non-Threshold) Risks

The equation for estimating "Incremental Lifetime Risk" from exposure to carcinogens (by compound and by pathway) is:

Risk = Cancer Potency Factor * ADD (life)

where:

Risk (Incremental Lifetime Risk)= Probability that person will manifest cancer, during lifetime, from the particular exposure condition;

ADD (life) = Average Daily Dose over a lifetime;

Cancer Potency Factor (CPF) for a compound = is the slope of the dose/response curve for cancers estimated

for the specific Chemical of Interest [(mg/kg/day)-1]; different CPF values may be estimated for ingestion and inhalation; such values are published by EPA's Cancer Assessment Group or have been estimated by other private or public organizations;

The "total" incremental lifetime cancer risk posed by a mixture of carcinogens is estimated as the sum of the risks for the individual chemicals.

A large number of calculations were made to estimate incremental risks of cancer. Included were all Compounds-of-Interest, all applicable exposure routes (ingestion, dermal contact, inhalation), the various receptor groups (workers involved in setting the foundation and in overall construction, pedestrian commuters passing by the site, and children playing near the site), and all three construction phases. Computer spreadsheets have been used to facilitate these calculations and the output is presented in Appendix B.

Summary results for total site risk are organized by receptor group and construction phase in Table 5-3 and in Figures 5-1 through 5-3. Shown on the figures is a dashed line at an incremental lifetime risk level (individual probability) of one in one hundred thousand (10^{-5}). This is a benchmark used in Massachusetts for judging the "significance" of estimated lifetime risks associated with the presence of chemicals in environmental media.

None of the total carcinogenic risks estimated for the proposed Parcel 18 construction project exceeded the 10^{-5} incremental lifetime risk level. Risks from carcinogenic effects to pedestrian commuters are estimated to be five orders of magnitude less than the MCP target level during all phases of work. Similarly, the estimated carcinogenic risks to children playing off-site is four orders of magnitude less than the target level. The estimated incremental lifetime carcinogenic risk to construction workers is five to 10 times less than the target level. The estimated carcinogenic risk to daycare children using the proposed play area is ten times less than the benchmark.

Based on the analysis presented in this report and the conservative assumptions employed, proposed construction on Parcel 18 and 18B is not considered to pose a significant incremental cancer risk to off-site receptors or to workers.

The exposure during proposed construction activities will be greater than exposure to trespassers under current site conditions or to office, maintenance, and emergency workers during post construction site conditions. Therefore, the risk under current or post construction conditions is also not considered to pose a

significant health risk to exposed receptors.

5.2.2 Estimation of Systemic (Threshold) Risks

The equation for estimating Hazard Index "Ratios" for exposure to non-carcinogens (by compound and by pathway) is:

$$\text{Ratio} = \text{ADD}_{\text{dur}}/\text{RfD}$$

where:

ADD_{dur} = Average Daily Dose over a selected period of exposure appropriate for the nature of risks being assessed (i.e., acute, subchronic, or chronic); in the present analysis the construction periods associated with individual phases (generally on the order of eighteen months) was used to assess potential chronic effects;

RfD = Reference dose used to assess the potential for chronic effects associated with extended periods (years to lifetime) of exposure; the Acceptable Intake Chronic (AIC) value is also used where an RfD value is not published.

In the absence of specific information on possible synergisms or antagonisms between or among chemicals, a total Hazard Index Ratio is estimated by summing the ratios for each compound. Individual or summed ratio values in excess of "1" are generally considered as "benchmarks" for indicating the potential for some (generally sublethal) health effects as a result of chronic (i.e., long-term) exposures. The MCP guidance identifies a benchmark of 0.2 (20% of the RfD) as appropriate for assessing the hazard associated with chemicals in environmental media. This lower target level is based on the presumption that individuals will be exposed to chemicals from other sources as part of everyday living and that as much as 80% of their exposure could come from these sources. Obviously, this presumption depends on the chemical under consideration and can be evaluated explicitly. However, in this analysis, results of calculations will be compared directly to the 0.2 benchmark value.

The convention of adding the hazards associated with various chemicals holds for doses that act on the same target organ/system or by the same mechanism. These assumptions and methods for estimating the "Ratio" and the "Risk" are conservative, i.e., protective of human health or environmental quality.

All calculations are presented in Appendix B. A summary of the total systemic health risks (i.e., ratios) is provided in Table 5-3 and the results are illustrated in Figures 5-4 through 5-6. These figures include a horizontal dashed line at a ratio of 0.2

corresponding with the MCP guidance that this level be used as a benchmark for evaluating systemic health risks.

All risks are below the 0.2 benchmark level and the proposed construction plan for the site is not considered to pose a "significant" health risk with regard to the potential for systemic health effects. Risks to off-site receptors (commuters and children at play) are typically one to two orders of magnitude below the benchmark level of 0.2 for total site risk.

The risks to construction workers directly involved in activities on-site, approach the 0.2 benchmark level. Considering the conservative assumptions used in this analysis and the fact that all risks are being evaluated as additive, it is unlikely that the actual risk ratios exceed the 0.2 benchmark level.

Examination of Appendix B reveals that the chemicals driving the risk to workers are two metals: thallium and lead. The sum of the hazard index ratios for these two metals accounts for 99.5% of the total hazard index ratio. Lead is present at the site at concentrations typical of urban background and is not considered to pose an unusual risk at this site. We have little information on the levels of thallium typically found in urban soils. The distribution of the compound over the site does not suggest the presence of waste "hot spots" but is rather uniform in both deep and shallow soils. Thallium emerges as a key contaminant at the site primarily because it has an extremely low RfD value. We note, that there is considerable uncertainty concerning the application of this RfD value to an environmental condition in which thallium (not otherwise specified) is present within a soil matrix.

The risk assessment assumes no dust control during construction. Thus, the estimated hazard index ratio is judged to be conservative. The only ratios that approached the 0.2 benchmark level are those involving construction workers. Appropriate dust control measures should ensure that exposures do not approach the benchmark value of 0.2 outlined in the MCP. The results of the analysis do not warrant any special remediation with regard to the presence of thallium or lead in soils on this site.

Since exposure to receptors under current or post construction conditions is estimated to be less than during construction, the risk to current or post construction receptors due to threshold effects will be less than the estimated risk to construction workers.

5.3 Uncertainty Analysis

Exposure Estimates

Sources of uncertainty in the exposure estimates include the following:

1. a number of assumptions, considered to be conservative, were made to develop estimates of dose to workers; the most important assumptions included estimates of the amount of soil incidentally ingested, inhaled, or coming into contact with skin; precautions taken to limit these exposure routes would greatly reduce any potential risks; as noted, dust control methods would be important here;

2. it was assumed that the same workers would be involved throughout the construction period; this is a reasonable assumption but may be conservative to the extent that different work crews would be involved in different aspects of the various construction efforts;

3. it was assumed that the bioavailability of metals from soil in the gut was 100%; this is a very conservative assumption inasmuch as many metals associated with soils will be part of the soil matrix and will pass through the gut in the matrix, unabsorbed; this is well known to occur and, in fact, analysis of metals in human wastes have been used to infer what the possible soil intake was; thus, the assumption that 100% of the ingested metals in soil are bioavailable may be conservative by one or more orders of magnitude; if this is the case for thallium in the soil, then all ratios would be considerably less than then benchmark value of 0.2;

4. it is assumed that future users of the site would not be at risk from site contaminants; this is based on the following considerations which are directly related to the proposed construction plans:

    - all construction will be on slab and there will be no sub-surface floors (basements) that could serve as receptacles for vapors known to occur at depth at the site;

    - after Phase I construction the eastern portion of the lot will be covered and landscaped or paved; there will not be an opportunity for future users of the buildings to come into direct contact with soils (other than topsoil) at this site.

Health Effects

Major sources of uncertainty with regard to health effects assessment include:

1. There are uncertainties associated with both the estimates of risk from carcinogens and non-carcinogens. The difficulty in extrapolating from data on high doses to animals to low doses in humans is well recognized and will not be discussed in detail here. However, the method used to estimate risks associated with exposure to carcinogenic PAH compounds is relatively new, and, therefore, is discussed here. In this assessment, the relative potency method developed by ICF Clement (1987) was used insofar as we derived CPF values for other carcinogenic PAH compounds. The existing CPF for B(a)P was used as the basis for the analysis. An alternative method would be to use the CPF for B(a)P as a surrogate for all other carcinogenic PAH compounds. It is widely recognized that this is an extremely conservative assumption. This is why we have used the relative potency approach but have maintained the existing CPF for B(a)P.

2. With regard to non-carcinogens, this report has used the Acceptable Intake Chronic number (AIC) or Risk Reference Dose (RfD) as benchmarks. These are threshold values which compensate for uncertainty through the application of safety factors. The AIC or RfD values used in this report were derived either by the EPA (published values) or consultants (e.g. Environ) by examining the various studies on the chemical and determining the No-Observed-Adverse-Effect-Level (NOAEL). However, the EPA (or consultants following EPA policy) do not simply use the NOAEL, but develop from it an AIC or RfD by dividing this NOAEL by a safety factor. The AIC or RfD has been viewed as the amount of a chemical to which a person can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering deleterious effects.

Generally, the safety factors used in deriving an AIC consist of multiples of 10. Each factor (of 10) represents a specific area of uncertainty inherent in the available data. For example, a safety factor may be developed by taking into account: 1) the expected differences in responsiveness between humans and animals (a factor of 10 applied here), 2) variability among individuals within the human population (a second factor of 10 applied here), and 3) a sparse data base (a third factor of 10). The result is that for many chemicals the AIC is calculated to be 100x less than the NOAEL and for some as much as 1000x less. In the case of the estimated AIC values for PAH compounds, this uncertainty level

(margin of safety) is on the order of 1000. Because of the margin of safety built into the AIC value, exceedence of the number has no immediate real meaning with regard to specific health effects, the frequency of effects, or the magnitude of effects. However, exceedence of the number should serve as a warning that the potential for unacceptable exposure does exist and precautions should be taken to limit exposure.

6.0 SAFETY, PUBLIC WELFARE, AND ENVIRONMENTAL RISK CHARACTERIZATION

The site is an urbanized area with no potentially impacted surface water body or other environmental receptors in the area. As indicated in Section 5, groundwater standards or guidelines are exceeded for heptachlor, lindane, and nickel. However, the groundwater in the area of the site is not a drinking water source.

The site does not presently pose any safety hazard. It is maintained as a fenced unoccupied area. It is not currently used for recreational or other purposes. Development of the site is not expected to pose a hazard to people living or working in the area due to site chemicals.

7.0 CONCLUSIONS AND RECOMMENDATIONS

We consider that the risks associated with current site conditions will be less than risks posed by construction on site. Therefore, we have not performed a quantitative assessment of current risk since such risks will be less than the risk to construction workers during the period of construction. Similarly, the exposure and hence risk to emergency, maintenance, and office workers in the foreseeable future is judged to be less than the risk to construction workers. Therefore, we have assessed risk to construction workers, offsite children, onsite (daycare) children, and pedestrian commuters.

Based upon the work described in this risk assessment, we conclude that:

> calculated risk due to carcinogenic and non-carcinogenic effects to the most exposed population (construction workers and daycare children) do not exceed DEQE benchmarks;
>
> noncarcinogenic risks to construction workers approach the DEQE benchmark level of 0.2 under scenarios which assume no dust control;
>
> the soil gas data indicate that the intrusion of vapors into above grade facilities is extremely unlikely.
>
> the risk to trespassers under current site conditions and office workers, emergency workers, and maintenance workers under future site conditions will be less than that to construction workers;
>
> risk to children using the area designated as a play area in future phases does not pose risks in excess of DEQE benchmarks.

We recommend:

> the use of dust control during construction activities;
>
> the removal of contaminated soils or the construction of a physical ground level barrier such as a cement cap between the soils and the proposed play area associated with the Phase III daycare center.

8.0 REFERENCES

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Tables

TABLE 2-1: GROUNDWATER CONTAMINANTS

CONTAMINANT	UNITS	RANGE	HITS/SAMPLE	DETECT. LIMIT
VOCS				
CHLOROBENZENE	UG/L	4.60	1/15	2
ACETONE	UG/L	11.50	1/15	5
TOLUENE	UG/L	3.4-92.4	2/15	2
ETHYL BENZENE	UG/L	10.1-620	4/15	2
XYLENES	UG/L	23.7-500	4/15	2
PESTICIDES				
ALPHA-BHC	UG/L	.3-30	8/12	.05
BETA-BHC	UG/L	.3-.55	8/12	.05
GAMMA-BHC	UG/L	.1-2	6/12	.05
HEPTACHLOR	UG/L	.1-.5	3/12	.05
ENDOSULPHAN II	UG/L	.7-1.0	2/12	.10
DIELDRIN	UG/L	.1	1/12	.10
ENDOSULFAN SULFATE	UG/L	.12-1.17	2/12	.10
4,4'-DDD	UG/L	.10	1/12	.10
METALS				
NICKEL	MG/L	.11-.23	5/5	.04
ZINC	MG/L	.006-.011	2/5	.005

TABLE 2-2: DEEP SOIL CONTAMINANTS

CONTAMINANT	UNITS	RANGE	HITS/SAMPLE	DETECT. LIMITS
VOC's				
CHLOROBENZENE	ppb	33-41	2/49	.3-3
ACETONE	ppb	34.4-210	2/49	.3-3
METHYLENE CHLORIDE	ppb	2.3-27.9	2/49	.3-3
1,2-DICHLOROETHANE	ppb	540	1/49	.3-3
CHLOROFORM	ppb	23	1/49	.3-3
1,1,2-TRICHLOROETHANE	ppb	41	1/49	.3-3
1,1,1-TRICHLOROETHANE	ppb	46-310	3/49	.3-3
BENZENE	ppb	3.2-49.5	2/49	.3-3
1,1,2,2-TETRACHLOROETHANE	ppb	77-84	2/49	.3-3
TOLUENE	ppb	23-34	2/49	.3-3
ETHYLBENZENE	ppb	17-6700	10/49	.3-3
TRICHLOROFLUOROMETHANE	ppb	41.1	1/49	.3-3
XYLENES	ppb	21.5-11000	12/49	.3-3
PAHS/CARCINOGENIC				
BENZO(A)PYRENE	ppm	.48-2.3	2/16	.30
BENZO(A)ANTHRACENE	ppm	1.7	1/16	.30
BENZO(B)FLUORANTHENE	ppm	3	1/16	.30
BENZO(K)FLUORANTHENE	ppm	3	1/16	.30
BENZO(G,H,I)PERYLENE	ppm	1.1	1/16	.30
CHRYSENE	ppm	.44-3.7	2/16	.30
DIBENZO(A,H)ANTHRACENE	ppm	.58	1/16	.30
INDENO(1,2,3-CD)PYRENE	ppm	.93	1/16	.30
PAHS/NON-CARCINOGENIC	ppm			
NAPHTHALENE	ppm	.43-4.1	3/16	.30
2-METHYLNAPHTHALENE	ppm	1.1-9.2	3/16	.30
ANTHRACENE	ppm	.97	1/16	.30
ACENAPTHENE	ppm	.52-.65	2/16	.30
FLUORENE	ppm	.42-.86	3/16	.30
PYRENE	ppm	2.7	1/16	.30
FLUORANTHENE	ppm	.72-6.3	2/16	.30
PHENANTHRENE	ppm	.61-5	4/16	.30
BIS(2-ETHYLHEXYL) PHTHALATE	ppm	1.1	1/16	.30
PESTICIDES				
4,4-DDT	ppb	56	1/49	20
ALPHA-BHC	ppb	18-400	12/49	10
BETA-BHC	ppb	15-1032	6/49	10
GAMMA-BHC	ppb	10-180	3/49	10
HEPTACHLOR	ppb	11.91-616	13/49	10
ALDRIN	ppb	15-167	4/49	10
HEPTACHLOR EPOXIDE	ppb	12-85	8/49	10
ENDOSULFAN I	ppb	11-110	5/49	10
DIELDRIN	ppb	40-114	4/49	20
4,4-DDE	ppb	19-50	5/49	20
ENDOSULFAN SULFATE	ppb	24-2080	2/49	20
4,4-DDD	ppb	33	1/49	20
METHOXYCHLOR	ppb	430	1/49	100

METALS				
ARSENIC	ppm	1.9-113	35/35	.10
BERYLLIUM	ppm	.1-.9	35/35	.25
CADMIUM	ppm	.3-1.0	23/35	.25
CHROMIUM	ppm	11-45	35/35	2.50
COPPER	ppm	10-375	35/35	1
LEAD	ppm	2.7-1460	31/35	2.50
MERCURY	ppm	.04-2.44	21/34	.10
NICKEL	ppm	8-49	35/35	2
SELENIUM	ppm	.1-1.2	9/35	.10
SILVER	ppm	.6-6	31/34	.50
THALLIUM	ppm	5.2-16	28/35	5
ZINC	ppm	.006-308	24/25	.25

TABLE 2-3: SURFACE SOIL CONTAMINANTS

CONTAMINANT	UNITS	RANGE	HITS/SAMPLE	DETECT. LIMIT
PAHS/CARCINOGENIC				
BENZO(A)PYRENE	ppm	1800-12000	5/11	330 and 660
BENZO(B)FLUORANTHENE	ppm	1800-7600	3/11	330 and 660
BENZO(K)FLUORANTHENE	ppm	1800-7600	3/11	330 and 660
CHRYSENE	ppm	1000-21000	9/11	330 and 660
PAHS/NON-CARCINOGENIC				
NAPHTHALENE	ppm	850-5800	2/11	330 and 660
ANTHRACENE	ppm	2000-75000	7/11	330 and 660
ACENAPTHENE	ppm	1300-6200	3/11	330 and 660
FLUORENE	ppm	1600-6400	2/11	330 and 660
PYRENE	ppm	1700-49000	8/11	330 and 660
PHENANTHRENE	ppm	2000-75000	8/11	330 and 660
OTHER BASE/NEUTRALS				
BIS(2-ETHYLHEXL) PHTHALATE	ppm	800-3900	7/11	330 and 660
DI-N-OCTYL PHTHALATE	ppm	1200-7100	2/11	330 and 660
DI-N-BUTYL PHTHALATE	ppm	2800	1/11	330 and 660
PESTICIDES				
4,4-DDT	ppb	154-241	2/11	20
ALDRIN	ppb	51-288	10/11	10
ENDOSULFAN I	ppb	72	1/11	10
4,4-DDE	ppb	67-76	2/11	20
AROCLOR	ppb	302	1/11	200
METALS				
ARSENIC	ppm	4.8-16	11/11	.1
BERYLLIUM	ppm	.5-.9	11/11	.25
CADMIUM	ppm	.3	1/11	.25
CHROMIUM	ppm	17-35	11/11	2.5
COPPER	ppm	14-60	11/11	1
LEAD	ppm	56-141	11/11	2.5
MERCURY	ppm	.2-3.4	11/11	.1
NICKEL	ppm	9-16	11/11	2
SELENIUM	ppm	.15-.31	8/11	.1
THALLIUM	ppm	7.5-11	11/11	5
ZINC	ppm	53-118	11/11	.25

TABLE 3-1 SOIL GAS SURVEY DATA SUMMARY

Compound	#hits/#stations	Range *	
Benzene	0/23	1	-
TCE	1/23	.50	12
Toluene	1/23	1	11
PCE	1/23	.50	3
Ethylbenzene	0/23	10	-
Xylene	0/23	2.50	-

* - lower end of range represents 0.5 times detection limit

TABLE 3-2

SOIL CONCENTRATIONS ASSOCIATED WITH EXCAVATION: PHASE I

Compounds	In Area of Excavation Mean Surface Composite (mg/kg)	Overall Average Concentration (mg/kg)	On-Site[**] Air Concentration (ug/m^3)
Carcinogenic PAH Compounds			
Benzo(a)pyrene	2.515	1.423	.0023
Benzo(a)anthracene	.330	.416	.0003
Benzo(b)fluoranthene	2.148	1.406	.0019
Benzo(k)fluoranthene	2.148	1.406	.0019
Benzo(g,h,i)perylene	.330	.378	.0003
Chrysene	3.683	2.238	.0033
Dibenzo(a,h)anthracene	.330	.346	.0003
Indeno(1,2,3-c,d)pyrene	.330	.368	.0003
Noncarcinogenic PAH Compounds			
Naphthalene(s)	.460	.401	.0004
Fluorene	.648	.509	.0006
Anthracene	5.540	2.975	.0050
Phenanthrene	6.033	3.529	.0054
Fluoranthene	.330	.703	.0003
Pyrene	5.308	2.988	.0048
Acenaphthene	.723	.538	.0007
Pesticides			
Aldrin	.095	.052	.0001
Dieldrin	.005	.005	.0000
Heptachlor	.005	.029	.0000
Heptachlor Epoxide	.005	.007	.0000
DDTR[*]	.099	.056	.0001
Endosulfan I	.010	.011	.0000
Endosulfan II	ND	0	
Endosulfan sulfate	.010	.066	.0000
Alpha-BHC	.005	.013	.0000
Beta-BHC	.005	.022	.0000
Gamma-BHC	.005	.005	.0000
Methoxychlor	ND	0	
Total Arochlors	ND	0	
Phthalate Esters			
Bis(2-ethylhexyl) phthalate	1.458	.894	.0013
Di-N-octyl phthalate	2.023	1.176	.0018
Di-N- butyl phthalate	.948	.639	.0009

TABLE 3-2
SOIL CONCENTRATIONS ASSOCIATED WITH EXCAVATION: PHASE I

	In Area of Excavation		
Compounds	Mean Surface Composite (mg/kg)	Overall Average Concentration (mg/kg)	On-Site** Air Concentration (ug/m^3)
Volatile Organic Compounds			
		20	
Benzene	VOCs		
Chlorobenzene	not detected		
Acetone	in	.017	
Methylene Chloride	soil gas	.006	
1,2 Dichloroethane	samples		
Chloroform	of		
Trichloroethane	vadose	.022	
1,1,2,2 Tetrachloroethane	zone		
Toluene	(low levels one sample)		
Ethylbenzene		.137	
Trichlorofluoromethane		.007	
Xylenes		.483	
Metals			
Arsenic	8.900	9.956	.0080
Lead	90	61.263	.0810
Mercury	.370	.321	.0003
Zinc	74	62.769	.0666
Copper	24.750	36.196	.0223
Silver	.250	1.139	.0002
Thallium	8.750	8.075	.0079

* sum of DDT, DDE, and DDD

** Air concentrations calculated using mean surface concentration

TABLE 3-3
SOIL CONCENTRATIONS ASSOCIATED WITH CONSTRUCTION: PHASE I
INCLUDES SURFACE SOILS IN ANTICIPATED STAGING AREA

Compounds	Staging Area Mean Surface Composite (mg/kg)	On-Site Air Concentration (ug/m^3)	Off-Site Air Concentration (ug/m^3)
Carcinogenic PAH Compounds			
Benzo(a)pyrene	6.165	.0055	.0016
Benzo(a)anthracene	.330	.0003	.0001
Benzo(b)fluoranthene	.330	.0003	.0001
Benzo(k)fluoranthene	.330	.0003	.0001
Benzo(g,h,i)perylene	.330	.0003	.0001
Chrysene	11	.0099	.0029
Dibenzo(a,h)anthracene	.330	.0003	.0001
Indeno(1,2,3-c,d)pyrene	.330	.0003	.0001
Noncarcinogenic PAH Compounds			
Naphthalene(s)	3.065	.0028	.0008
Fluorene	3.365	.0030	.0009
Anthracene	37.665	.0339	.0098
Phenanthrene	38.950	.0351	.0101
Fluoranthene	.330	.0003	.0001
Pyrene	26.550	.0239	.0069
Acenaphthene	3.265	.0029	.0008
Pesticides			
Aldrin	.190	.00017	.00005
Dieldrin	.005	.000005	.000001
Heptachlor	.005	.000005	.000001
Heptachlor Epoxide	.005	.000005	.000001
DDTR*	.010	.000009	.000003
Endosulfan I	.039	.00003	.00001
Endosulfan II	ND		
Endosulfan sulfate	.010	.00001	.000003
Alpha-BHC	.005	.000005	.000001
Beta-BHC	.005	.000005	.000001
Gamma-BHC	.005	.000005	.000001
Methoxychlor	ND		
Total Arochlors	ND		
Phthalate Esters			
Bis(2-ethylhexyl) phthalate	.815	.000734	.000212
Di-N-octyl phthalate	ND		
Di-N- butyl phthalate	ND		

TABLE 3-3

TABLE 3-3

SOIL CONCENTRATIONS ASSOCIATED WITH CONSTRUCTION: PHASE I
INCLUDES SURFACE SOILS IN ANTICIPATED STAGING AREA

Compounds	Staging Area Mean Surface Composite (mg/kg)	On-Site Air Concentration (ug/m^3)	Off-Site Air Concentration (ug/m^3)
Volatile Organic Compounds			
Benzene	VOCs not detected in soil gas samples of vadose zone (low levels in one sample)		
Chlorobenzene			
Acetone			
Methylene Chloride			
1,2 Dichloroethane			
Chloroform			
Trichloroethane			
1,1,2,2 Tetrachloroethane			
Toluene			
Ethylbenzene			
Trichlorofluoromethane			
Xylenes			
Metals			
Arsenic	6.700	.0060	.0017
Lead	89.500	.0806	.0233
Mercury	.415	.0004	.0001
Zinc	80	.0720	.0208
Copper	21.500	.0194	.0056
Silver	.250	.0002	.0001
Thallium	8.750	.0079	.0023

* sum of DDT, DDE, and DDD

TABLE 3-4
SOIL CONCENTRATIONS ASSOCIATED WITH EXCAVATION: PHASE II

	In Area of Excavation	
Compounds	Mean Surface Composite (mg/kg)	Overall Average Concentration (mg/kg)
Carcinogenic PAH Compounds		
Benzo(a)pyrene	6.165	3.285
Benzo(a)anthracene	.330	.413
Benzo(b)fluoranthene	.330	.413
Benzo(k)fluoranthene	.330	.413
Benzo(g,h,i)perylene	ND	
Chrysene	11	5.693
Dibenzo(a,h)anthracene	ND	
Indeno(1,2,3-c,d)pyrene	ND	
Noncarcinogenic PAH Compounds		
Naphthalene(s)	3.065	3.040
Fluorene	3.365	1.980
Anthracene	37.665	18.998
Phenanthrene	38.950	20.008
Fluoranthene	.330	.428
Pyrene	26.550	13.523
Acenaphthene	3.265	1.878
Pesticides		
Aldrin	.190	.097
Dieldrin	.005	.009
Heptachlor	.005	.013
Heptachlor Epoxide	.005	.010
DDTR*	.010	.012
Endosulfan I	.039	.029
Endosulfan II	ND	
Endosulfan sulfate	.010	.008
Alpha-BHC	.005	.012
Beta-BHC	.005	.069
Gamma-BHC	.005	.008
Methoxychlor	ND	
Total Arochlors	ND	
Phthalate Esters		
Bis(2-ethylhexyl) phthalate	.815	.573
Di-N-octyl phthalate	ND	
Di-N- butyl phthalate	ND	

TABLE 3-5

SOIL CONCENTRATIONS ASSOCIATED WITH CONSTRUCTION: PHASES II AND III
THESE SURFACE SOILS ARE LOCATED IN STAGING AREA

Compounds	Staging Area Mean Surface Composite (mg/kg)	On-Site Air Concentration (ug/m^3)	Off-Site Air Concentration (ug/m^3)
Carcinogenic PAH Compounds			
Benzo(a)pyrene	1.198	.0011	.0005
Benzo(a)anthracene	.330	.0003	.0001
Benzo(b)fluoranthene	1.418	.0013	.0006
Benzo(k)fluoranthene	1.418	.0013	.0006
Benzo(g,h,i)perylene	.330	.0003	.0001
Chrysene	2.326	.0021	.0009
Dibenzo(a,h)anthracene	ND		
Indeno(1,2,3-c,d)pyrene	ND		
Noncarcinogenic PAH Compounds			
Naphthalene(s)	.330	.0003	.0001
Fluorene	.584	.0005	.0002
Anthracene	5.126	.0046	.0021
Phenanthrene	5.126	.0046	.0021
Fluoranthene	.330	.0003	.0001
Pyrene	4.006	.0036	.0016
Acenaphthene	.524	.0005	.0002
Pesticides			
Aldrin	.083	.0001	.000033
Dieldrin	.005	.000005	.000002
Heptachlor	.005	.000005	.000002
Heptachlor Epoxide	.005	.000005	.000002
DDTR*	.039	.000035	.000016
Endosulfan I	.005	.000005	.000002
Endosulfan II	.005	.000005	.000002
Endosulfan sulfate	.005	.000005	.000002
Alpha-BHC	.005	.000005	.000002
Beta-BHC	.005	.000005	.000002
Gamma-BHC	.005	.000005	.000002
Methoxychlor	ND		
Total Arochlors	.140	.0001	.0001
Phthalate Esters			
Bis(2-ethylhexyl) phthalate	ND		
Di-N-octyl phthalate	.504	.0005	.000202
Di-N-butyl phthalate	ND		

TABLE 3-5
SOIL CONCENTRATIONS ASSOCIATED WITH CONSTRUCTION: PHASES II AND III
THESE SURFACE SOILS ARE LOCATED IN STAGING AREA

Compounds	Staging Area Mean Surface Composite (mg/kg)	On-Site Air Concentration (ug/m^3)	Off-Site Air Concentration (ug/m^3)
Volatile Organic Compounds			
Benzene	VOCs not detected in soil gas samples of vadose zone (low level one sample)		
Chlorobenzene			
Acetone			
Methylene Chloride			
1,2 Dichloroethane			
Chloroform			
Trichloroethane			
1,1,2,2 Tetrachloroethane			
Toluene			
Ethylbenzene			
Trichlorofluoromethane			
Xylenes			
Metals			
Arsenic	9.400	.0085	.0038
Lead	115	.1035	.0460
Mercury	.050	.00005	.00002
Zinc	118	.1062	.0472
Copper	25	.0225	.0100
Silver	1.300	.0012	.0005
Thallium	9.700	.0087	.0039

* sum of DDT, DDE, and DDD

TABLE 3-6
SOIL CONCENTRATIONS ASSOCIATED WITH EXCAVATION: PHASE III

	In Area of Excavation		In Proposed Play Area
Compounds	Mean Surface Composite (mg/kg)	Overall Average Concentration (mg/kg)	Average Concentration (mg/kg)
Carcinogenic PAH Compounds			
Benzo(a)pyrene	1.198	.764	1.80
Benzo(a)anthracene	.330	.330	ND
Benzo(b)fluoranthene	1.418	.874	1.80
Benzo(k)fluoranthene	1.418	.874	1.80
Benzo(g,h,i)perylene	.330	.330	ND
Chrysene	2.326	1.369	2.80
Dibenzo(a,h)anthracene	ND	.330	ND
Indeno(1,2,3-c,d)pyrene	ND	.413	ND
Noncarcinogenic PAH Compounds			
Naphthalene(s)	.330	1.951	ND
Fluorene	.584	.498	ND
Anthracene	5.126	2.728	6.10
Phenanthrene	5.126	2.804	6.10
Fluoranthene	.330	.371	ND
Pyrene	4.006	2.209	5.60
Acenaphthene	.524	.427	ND
Pesticides			
Aldrin	.083	.051	.13
Dieldrin	.005	.009	ND
Heptachlor	.005	.025	ND
Heptachlor Epoxide	.005	.008	ND
DDTR*	.039	.023	ND
Endosulfan I	.005	.008	ND
Endosulfan II	.005		ND
Endosulfan sulfate	.005		ND
Alpha-BHC	.005	.024	ND
Beta-BHC	.005	.006	ND
Gamma-BHC	.005	.013	ND
Methoxychlor	ND	.026	ND
Total Arochlors	.140	.120	.30
Phthalate Esters			
Bis(2-ethylhexyl) phthalate	.330	.426	ND
Di-N-octyl phthalate	.504	.417	ND
Di-N- butyl phthalate	ND		ND

TABLE 3-6
SOIL CONCENTRATIONS ASSOCIATED WITH EXCAVATION: PHASE III

	In Area of Excavation		In Proposed Play Area
Compounds	Mean Surface Composite (mg/kg)	Overall Average Concentration (mg/kg)	Average Concentration (mg/kg)
Volatile Organic Compounds			
Benzene	VOCs	.004	VOCs
Chlorobenzene	not detected	.059	not detected
Acetone	in		in
Methylene Chloride	soil gas		soil gas
1,2 Dichloroethane	samples		samples
Chloroform	of		of
1,1,1 Trichloroethane	vadose	.007	vadose
1,1,2,2 Tetrachloroethane	zone	.009	zone
Toluene	(low levels one sample)	.006	
Ethylbenzene		.579	
Trichlorofluoromethane			
Xylenes		.980	
Metals			
Arsenic	9.400	8.454	4.80
Lead	115	113.204	75
Mercury	.050	.224	.47
Zinc	118	74	71
Copper	25	21	60
Silver	1.300	1.371	ND
Thallium	9.700	8.286	9

* sum of DDT, DDE, and DDD

TABLE 3-7
SCENARIO I: PHASE I EXPOSED CONSTRUCTION WORKER DURING FOUNDATION CONSTRUCTION
PATHWAYS: DERMAL CONTACT; INCIDENTAL INGESTION; AND INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposed Skin Area (1)	3,570 cm2	Anderson et al., 1985
Exposure Duration	60 days	Construction Plan
Daily Exposure Period	10 hours	Estimated
Skin Soil Coverage	1mg/cm2-day	Hawley, J.K., 1985
Dermal Absorption Factor		
Semi-volatiles	0.05	EPA,1989
Volatiles	0.5	EPA,1989
Ingestion Rate	100 mg/day	EPA,1989
Oral Absorption Factor		EPA,1989
Semi-volatiles	0.3	
Volatiles	1.0	
Inorganics	1.0	
Lead	0.3	
Inhalation Rate	2 m3/hr	Anderson et al., 1985
Inhalation Absorption Factor		
Volatiles	1.0	EPA,1989
Semi-volatiles	1.0	EPA,1989
Inorganics	1.0	EPA,1989
Exposure Point Concentration		Table 3-2

Notes: (1) represents neck, arms, and hands

TABLE 3-8
SCENARIO I: PHASE I EXPOSED CONSTRUCTION WORKER
EXPOSURE FROM STAGING AREA DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION AND SOIL INGESTION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	10 hours	Estimated
PM-10 Airborne Dust	90 ug/m3	(2)
Absorption Factor Inhalation		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	2.0 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-3
Ingestion Rate	50 mg/day	Estimated
Absorption Factor Ingestion (see Table 3-7)		

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988)

TABLE 3-9
SCENARIO II: PHASE I EXPOSED PEDESTRIAN COMMUTER
EXPOSURE DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	20 minutes/day	Estimated
PM-10 Airborne Dust	90 ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	1.25 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-3

(1) based on 18 months construction and 5 day work week
(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988)

TABLE 3-10

SCENARIO III: PHASE I EXPOSED CHILDREN OFF SITE
EXPOSURE DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	5-18 years	Assumed
Average Body Weight	40 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	4 hours/day	Estimated
PM-10 Airborne Dust	26ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	1.25 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-3

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988) modelled particle concentrations at 150 meters from site

TABLE 3-11

SCENARIO IV: PHASE II EXPOSED CONSTRUCTION WORKER
EXPOSURE DURING FOUNDATION CONSTRUCTION
PATHWAYS: DERMAL CONTACT; INCIDENTAL INGESTION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposed Skin Area (1)	3,570 cm2	Anderson et al., 1985
Exposure Duration	60 days	Construction Plan
Daily Exposure Period	10 hours	Estimated
Skin Soil Coverage	1mg/cm2-day	Hawley, J.K., 1985
Dermal Absorption Factor		
Semi-volatiles	0.05	EPA,1989
Volatiles	0.5	EPA,1989
Ingestion Rate	100 mg/day	EPA,1989
Oral Absorption Factor		EPA,1989
Semi-volatiles	0.3	
Volatiles	1.0	
Inorganics	1.0	
Lead	0.3	
Exposure Point Concentration		Table 3-4

Notes: (1) represents neck, arms, and hands

TABLE 3-12
SCENARIO IV: PHASE II EXPOSED CONSTRUCTION WORKER DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	10 hours	Estimated
PM-10 Airborne Dust	90 ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	2.0 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-5

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988)

TABLE 3-13
SCENARIO IV: PHASE II EXPOSED CONSTRUCTION WORKER
STAGING AREA SOILS
PATHWAYS: INCIDENTAL INGESTION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposed Skin Area (1)	3,570 cm2	Anderson et al., 1985
Exposure Duration	360 days	Construction Plan
Daily Exposure Period	10 hours	Estimated
Skin Soil Coverage	1mg/cm2-day	Hawley, J.K., 1985
Dermal Absorption Factor		
Semi-volatiles	0.05	EPA,1989
Volatiles	0.5	EPA,1989
Ingestion Rate	50 mg/day	Estimated
Oral Absorption Factor		EPA,1989
Semi-volatiles	0.3	
Volatiles	1.0	
Inorganics	1.0	
Lead	0.3	
Exposure Point Concentration		Table 3-5

Notes: (1) represents neck, arms, and hands

TABLE 3-14
SCENARIO V: PHASE II EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	20 minutes/day	Estimated
PM-10 Airborne Dust	90 ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	1.25 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-5

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988)

TABLE 3-15
SCENARIO VI: PHASE II EXPOSED CHILDREN OFF SITE DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	5-18 years	Assumed
Average Body Weight	40 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	4 hours/day	Estimated
PM-10 Airborne Dust	40 ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	1.25 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-5

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988) modelled particle concentrations at 75 meters from site

TABLE 3-16
SCENARIO VII: PHASE III EXPOSED CONSTRUCTION WORKER
EXPOSURE DURING FOUNDATION CONSTRUCTION
PATHWAYS: DERMAL CONTACT; INCIDENTAL INGESTION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposed Skin Area (1)	3,570 cm2	Anderson et al., 1985
Exposure Duration	60 days	Construction Plan
Daily Exposure Period	10 hours	Estimated
Skin Soil Coverage	1mg/cm2-day	Hawley, J.K., 1985
Dermal Absorption Factor		
Semi-volatiles	0.05	EPA,1989
Volatiles	0.5	EPA,1989
Ingestion Rate	100 mg/day	EPA,1989
Oral Absorption Factor		EPA,1989
Semi-volatiles	0.3	
Volatiles	1.0	
Inorganics	1.0	
Lead	0.3	
Exposure Point Concentration		Table 3-6

Notes: (1) represents neck, arms, and hands

TABLE 3-17

SCENARIO VII: PHASE III EXPOSED CONSTRUCTION WORKER
EXPOSURE FROM STAGING AREA SOILS
PATHWAYS: INCIDENTAL INGESTION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposed Skin Area (1)	3,570 cm2	Anderson et al., 1985
Exposure Duration	720 days	Construction Plan
Daily Exposure Period	10 hours	Estimated
Skin Soil Coverage	1mg/cm2-day	Hawley, J.K., 1985
Dermal Absorption Factor		
Semi-volatiles	0.05	EPA,1989
Volatiles	0.5	EPA,1989
Ingestion Rate	50 mg/day	Estimated
Oral Absorption Factor		EPA,1989
Semi-volatiles	0.3	
Volatiles	1.0	
Inorganics	1.0	
Lead	0.3	
Exposure Point Concentration		Table 3-5

Notes: (1) represents neck, arms, and hands

TABLE 3-18

SCENARIO VII: PHASE III EXPOSED CONSTRUCTION WORKER
EXPOSURE FROM STAGING AREA
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	720 days (1)	Construction Plan
Daily Exposure Period	10 hours	Estimated
PM-10 Airborne Dust	90 ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	2.0 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-5

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988)

TABLE 3-19
SCENARIO VIII: PHASE III EXPOSED PEDESTRIAN COMMUTER EXPOSURE DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	18-65 years	Assumed
Average Body Weight	70 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	720 days (1)	Construction Plan
Daily Exposure Period	20 minutes/day	Estimated
PM-10 Airborne Dust	90 ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	1.25 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-5

(1) based on 18 months construction and 5 day work week
(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988)

TABLE 3-20
SCENARIO IX: PHASE III EXPOSED CHILDREN OFF SITE
EXPOSURE DURING BUILDING CONSTRUCTION
PATHWAY: INHALATION

PARAMETER	ASSUMED VALUE	REFERENCE
Age	5-18 years	Assumed
Average Body Weight	40 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposure Duration	360 days (1)	Construction Plan
Daily Exposure Period	4 hours/day	Estimated
PM-10 Airborne Dust	40ug/m3	(2)
Absorption Factor		
Semi-volatiles	1.0	Estimated Worst Case
Volatiles	1.0	Estimated Worst Case
Inhalation Rate	1.25 m3/hr	Anderson et al., 1985
Exposure Point Concentration		Table 3-5

(1) based on 18 months construction and 5 day work week

(2) based on average 180 ug/m3 at average construction site (Trans. Res. Bd., 1978) and estimated 50% of airborne particles less than 10 microns (EPA, 1988) modelled particle concentrations at 75 meters from site

TABLE 3-21
SCENARIO X: EXPOSED DAYCARE CHILDREN IN PROPOSED PLAYGROUND DURING POST CONSTRUCTION
PATHWAYS: DERMAL CONTACT; INCIDENTAL INGESTION;

PARAMETER	ASSUMED VALUE	REFERENCE
Age	1-6 years	Assumed
Average Body Weight	40 kilograms	Anderson et al., 1985
Average Lifetime	70 years	Assumed
Exposed Skin Area (1)	1.030 cm2	DEQE, 1989
Exposure Duration	140 days	Estimated
Daily Exposure Period	4 hours	Estimated
Skin Soil Coverage	1mg/cm2-day	Hawley, J.K., 1985
Dermal Absorption Factor		
Semi-volatiles	0.05	EPA,1989
Volatiles	0.5	EPA,1989
Ingestion Rate	100 mg/day	DEQE,1989
Oral Absorption Factor		EPA,1989
Semi-volatiles	0.3	
Volatiles	1.0	
Inorganics	1.0	
Lead	0.3	
Exposure Point Concentration		Table 3-6

Notes: (1) represents arms, and hands

TABLE 4-1

EPA WEIGHT-OF-EVIDENCE CATEGORIES FOR POTENTIAL CARCINOGENS

EPA Category	Description of Group	Description of Evidence
Group A	Human Carcinogen	Sufficient evidence from epidemiology studies to support a causal association between exposure and human cancer.
Group B1	Probable Human Carcinogen	Limited evidence of carcinogenicity in humans from epidemiology studies.
Group B2	Probable Human Carcinogen	Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans.
Group C	Possible Human Carcinogen	Limited evidence of carcinogenicity in animals; no data for humans.
Group D	Not Classified	Inadequate evidence of carcinogenicity in animals.
Group E.	No Evidence of Carcinogenicity in Humans	No evidence for carcinogenicity in at least two adequate animal tests or in both epidemiology and animal studies

TABLE 4-2

COMPOUNDS-OF-INTEREST EVALUATED AS POTENTIAL CARCINOGENS AND ASSOCIATED EPA WEIGHT OF EVIDENCE (SEE TABLE 4-1)

Chemicals	EPA Weight of Evidence	Exposure Route
Potentially Carcinogenic PAH Compounds		
Benzo(a)anthracene	Probable Human Carcinogen (B2)	oral/inhal
Chrysene	Probable Human Carcinogen (B2)	oral/inhal
Benzo(b)fluoranthene	Probable Human Carcinogen (B2)	oral/inhal
Benzo(k)fluoranthene	Not Classified	oral/inhal
Benzo(a)pyrene	Probable Human Carcinogen (B2)	oral/inhal
Indeno(1,2,3-cd)pyrene	Probable Human Carcinogen (B2)	oral/inhal
Dibenzo(a,h)anthracene	Probable Human Carcinogen (B2)	oral/inhal
Benzo(g,h,i)perylene	Probable Human Carcinogen (B2)	oral/inhal
Pesticides		
Aldrin	Probable Human Carcinogen (B2)	oral/inhal
DDTR	Probable Human Carcinogen (B2)	oral/inhal
Dieldrin	Probable Human Carcinogen (B2)	oral/inhal
Heptachlor	Probable Human Carcinogen (B2)	oral/inhal
Heptachlor Epoxide	Probable Human Carcinogen (B2)	oral/inhal
BHC compounds	Probable Human Carcinogen (B2)	oral/inhal
Phthalate Esters		
Bis(2-ethyl hexyl) phthalate	Probable Human Carcinogen (B2)	oral/inhal
Volatile Organic Compounds		
Benzene	Human Carcinogen (A)	oral/inhal
Methylene Chloride	Probable Human Carcinogen (B2)	oral/inhal
1,2 Dichloroethane	Probable Human Carcinogen (B2)	oral/inhal
Chloroform	Probable Human Carcinogen (B2)	oral/inhal
1,1,2,2 Tetrachloroethane	Possible Carcinogen (C)	oral/inhal
1,1,2 Trichloroethane	Possible Carcinogen (C)	oral/inhal
Metals		
Arsenic	Human Carcinogen (A) Note: oral route is under review	oral/inhal
Lead	Probable Human Carcinogen (B2) - still under review	

TABLE 4-3

RELATIVE POTENCIES OF PAH COMPOUNDS

PAH Chemical	Relative Potency	Study Reference
benzo(a)pyrene	1.0	
benz(a)anthracene	0.145	Bingham and Falk (1969)
benzo(b)fluoranthene	0.140	Deutsch-Wenzel et al. (1983)
benzo(k)fluoranthene	0.066	Deutsch-Wenzel et al. (1983)
benzo(ghi)perylene	0.022	Deutsch-Wenzel et al. (1983)
chrysene	0.0044	Wynder and Hoffmann (1959)
dibenz(ah)anthracene	1.11	Wynder and Hoffmann (1959)
indeno(1,2,3-cd)pyrene	0.232	Deutsch-Wenzel et al. (1983)

TABLE 4-4
CANCER POTENCY FACTORS FOR COMPOUNDS OF INTEREST

Compounds	Relative Potency Factor	Oral CPF (mg/kg/day)$^{-1}$	Inhalation CPF (mg/kg/day)$^{-1}$	Data Source
Carcinogenic PAH Compounds*				
Benzo(a)pyrene	1	11.500	6.100	EPA WQC
Benzo(a)anthracene	.145	1.668	.885	Estimated
Benzo(b)fluoranthene	.140	1.610	.854	Estimated
Benzo(k)fluoranthene	.066	.759	.403	Estimated
Benzo(g,h,i)perylene	.022	.253	.134	Estimated
Chrysene	.004	.046	.024	Estimated
Dibenzo(a,h)anthracene	1.110	12.765	6.771	Estimated
Indeno(1,2,3-c,d)pyrene	.232	2.668	1.415	Estimated
Noncarcinogenic PAH Compounds*				
Naphthalene(s)		NA	NA	
Fluorene		NA	NA	
Anthracene		NA	NA	
Phenanthrene		NA	NA	
Fluoranthene		NA	NA	
Pyrene		NA	NA	
Acenaphthene		NA	NA	
Pesticides				
Aldrin		17	17	EPA IRIS
Dieldrin		16	16	EPA IRIS
Heptachlor		4.500	4.500	EPA IRIS
Heptachlor Epoxide		9.100	9.100	EPA IRIS
DDTR*		.340	.340	EPA IRIS
Endosulfan I		NA	NA	
Endosulfan II		NA	NA	
Endosulfan sulfate		NA	NA	
Alpha-BHC		6.300	6.300	EPA IRIS
Beta-BHC		1.800	1.800	EPA IRIS
Gamma-BHC		1.300	1.300	EPA IRIS
Methoxychlor		NA	NA	
Total Arochlors		7.700	7.700	EPA IRIS
Phthalate Esters				
Bis(2-ethylhexyl) phthalate		1.400E-2	1.400E-2	EPA IRIS
Di-N-octyl phthalate		NA	NA	
Di-N- butyl phthalate		NA	NA	

TABLE 4-4
CANCER POTENCY FACTORS FOR COMPOUNDS OF INTEREST

Compounds	Relative Potency Factor	Oral CPF $(mg/kg/day)^{-1}$	Inhalation CPF $(mg/kg/day)^{-1}$	Data Source
Volatile Organic Compounds				
Benzene		.052	.026	EPA IRIS
Chlorobenzene		NA	NA	
Acetone		NA	NA	
Methylene Chloride		.008	.014	EPA IRIS
1,2 Dichloroethane		.091	.035	EPA IRIS
Chloroform		.006	.081	EPA IRIS
Trichloroethane		.057	.057	EPA IRIS
1,1,2,2 Tetrachloroethane		.200	.200	EPA IRIS
Toluene		NA	NA	
Ethylbenzene		NA	NA	
Trichlorofluoromethane		NA	NA	
Xylenes		NA	NA	
Metals				
Arsenic		1.500	5	EPA IRIS
Lead		NA	NA	
Mercury		NA	NA	
Zinc		NA	NA	
Copper		NA	NA	
Silver		NA	NA	
Thallium		NA	NA	

TABLE 4-5

ESTIMATED AIC VALUES FOR PAH COMPOUNDS (ENVIRON, 1986)

Chemical	Estimated AIC Value	Basis For Number	Target Organ(s)	Observed Effect	Uncertainty Factor
Acenaphthene	0.2 mg/kg/day	LOAEL for rats; oral dose of 1.0 g/kg/day and higher dose of 2.0 g/kg/day (Knobloch et al. 1969)	Liver Kidney Bronchi	Gain in liver weight; increase in enzyme activity; increased urine flow; morphological damage to kidney and liver and mild bronchitis at higher dose.	5000 includes 1000 to account for intra and inter species variability and a factor of 5 to compensate for use of LOAEL rather than
Naphthalene	0.053 mg/kg/day	Subchronic NOAEL for mice of 53 mg/kg/day (Shopp et al. 1984) administered by gavage	Spleen Lung Thymus Body Wt.	Decreased Spleen and Thymus Weight; Increased Lung Weight	1000 accounts for intra and inter-species variability
Naphthalene	0.0053 mg/kg/day	NOAEL for mice of 5.3 mg/kg/day (Shopp et al. 1984) administered by gavage	Serum Protein Levels	Increase in Serum Protein Levels	1000 accounts for intra and inter-species variability
Acenaphthylene	0.03 mg/kg/day	Subchronic LOAEL for rats of 300 mg/kg/day as an oral dose	Body Wt. Liver Kidney	Decrease in organ weight; minor degeneration; increased enzyme activity; increased urine flow; study considered inadequate	10,000 1000 accounts for intra and inter-species differences; additional 10 accounts for use of LOAEL and method problems
Pyrene	0.06 mg/kg/day	Subchronic LOAEL based on oral dose of 120 mg/kg/day (White and White, 1939)	Liver	Slightly fatty and enlarged liver	2000 1000 accounts for intra and inter-species differences; additional 2 accounts for use of LOAEL

TABLE 4-6
REFERENCE DOSE (RfD) OR ACCEPTABLE INTAKE CHRONIC (AIC) VALUES FOR COMPOUNDS OF INTEREST

Compounds	Oral RfD/AIC (mg/kg/day)	Inhalation RfD/AIC (mg/kg/day)	Comments
Carcinogenic PAH Compounds*			
Benzo(a)pyrene	.010	.010	These are
Benzo(a)anthracene	.010	.010	all estimated
Benzo(b)fluoranthene	.010	.010	based on
Benzo(k)fluoranthene	.010	.010	limited data
Benzo(g,h,i)perylene	.010	.010	presented in
Chrysene	.010	.010	Environ (1986)
Dibenzo(a,h)anthracene	.010	.010	
Indeno(1,2,3-c,d)pyrene	.010	.010	
Noncarcinogenic PAH Compounds*			
Naphthalene	.005	.005	<--- See Text
Fluorene	.010	.010	estimated
Anthracene	.010	.010	estimated
Phenanthrene	.010	.010	estimated
Fluoranthene	.010	.010	estimated
Pyrene	.010	.010	estimated
Acenaphthene	.010	.010	estimated
Pesticides			
Aldrin	3.000E-5	3.000E-5	EPA IRIS
Dieldrin	5.000E-5	5.000E-5	EPA IRIS
Heptachlor	3.000E-4	3.000E-4	EPA IRIS
Heptachlor Epoxide	1.300E-5	1.300E-5	EPA IRIS
DDTR*	5.000E-4	5.000E-4	Based on DDT
Endosulfan I	5.000E-5	5.000E-5	EPA IRIS
Endosulfan II	5.000E-5	5.000E-5	Based on Endosulfan I
Endosulfan sulfate	5.000E-5	5.000E-5	Based on Endosulfan I
Alpha-BHC	3.000E-4	3.000E-4	Based on Gamma-BHC
Beta-BHC	3.000E-4	3.000E-4	Based on Gamma-BHC
Gamma-BHC	3.000E-4	3.000E-4	EPA IRIS
Methoxychlor	.050	.050	EPA IRIS
Total Arochlors	1.000E-4	1.000E-4	EPA IRIS
Phthalate Esters			
Bis(2-ethylhexyl) phthalate	.020	.020	EPA PHRED Data Base
Di-N-octyl phthalate	.020	.020	Based on DEHP
Di-N- butyl phthalate	.100	.100	EPA IRIS

TABLE 4-6

REFERENCE DOSE (RfD) OR ACCEPTABLE INTAKE CHRONIC (AIC) VALUES FOR COMPOUNDS OF INTEREST

Compounds	Oral RfD/AIC (mg/kg/day)	Inhalation RfD/AIC (mg/kg/day)	Comments
Volatile Organic Compounds			
Benzene	7.000E-4	7.000E-4	proposed RfD
Chlorobenzene	.005	.005	Based on inhalation
Acetone	NA	NA	
Methylene Chloride	.060	.060	EPA IRIS
1,2 Dichloroethane	.074	.074	Estimated from advisory
Chloroform	.010	.010	EPA IRIS
Trichloroethane	.004	.004	EPA IRIS
1,1,2,2 Tetrachloroethane	NA	NA	
Toluene	.300	1.500	EPA IRIS
Ethylbenzene	.100	.100	EPA IRIS
Trichlorofluoromethane	NA	NA	
Xylenes	2	2	EPA IRIS
Metals			
Arsenic	.001	.001	EPA IRIS
Lead	5.000E-4	5.000E-4	Estimated from MCL
Mercury	3.000E-4	3.000E-4	EPA IRIS
Zinc	.210	.010	EPA IRIS
Copper	.037	.010	EPA IRIS
Silver	.003	.003	EPA IRIS
Thallium	7.000E-5	7.000E-5	EPA IRIS

* AIC values of 0.01 mg/kg/day are believed to be conservative estimates based on the limited available information

TABLE 5-1
APPLICABLE STANDARDS AND GUIDELINES FOR GROUND WATER IN COMPARISON TO LEVELS AT THE PARCEL 18 SITE

Compounds	STANDARD, CRITERION, OR GUIDELINE GROUNDWATER ug/l	REFERENCE	SITE CONDITIONS PARCEL 18 GROUNDWATER
Carcinogenic PAH Compounds	0.028 (1)	EPA WQC	Undetected
Benzo(a)pyrene		Based on 10^{-5}	Undetected
Benzo(a)anthracene		Lifetime Risk	Undetected
Benzo(b)fluoranthene		of Drinking	Undetected
Benzo(k)fluoranthene		Water	Undetected
Benzo(g,h,i)perylene			Undetected
Chrysene			Undetected
Dibenzo(a,h)anthracene			Undetected
Indeno(1,2,3-c,d)pyrene			Undetected
Noncarcinogenic PAH Compounds			Undetected
Naphthalene	none		Undetected
Fluorene	none		Undetected
Anthracene	none		Undetected
Phenanthrene	none		Undetected
Fluoranthene	42	EPA WQC	Undetected
Pyrene	none		Undetected
Acenaphthene	none		Undetected
Pesticides			
Aldrin	.740	EPA WQC (1)	Undetected
Dieldrin	.710	EPA WQC (1)	Max Value = 0.100
Heptachlor	.400	MCL	Max value = 0.500
Heptachlor Epoxide	.200	MCL	Undetected
DDTR*	.240	EPA WQC (1)	Max Value = 0.100
Endosulfan I	74	EPA WQC	Undetected
Endosulfan II	74	EPA WQC	Max Value = 1
Endosulfan sulfate	74	EPA WQC	Max Value = 1.17
Alpha-BHC	.200	(2)	Max Value = 3
Beta-BHC	.200	(2)	Max Value = 0.55
Gamma-BHC	.200	MCL	Max Value = 2
Methoxychlor	400	MCL	Undetected
Total Arochlors	.500	MCL	Undetected

TABLE 5-1

APPLICABLE STANDARDS AND GUIDELINES FOR GROUND WATER IN COMPARISON TO LEVELS AT THE PARCEL 18 SITE

Compounds	STANDARD, CRITERION, OR GUIDELINE GROUNDWATER ug/l	REFERENCE	SITE CONDITIONS PARCEL 18 GROUNDWATER
Volatile Organic Compounds			
Benzene	5	MCL	Undetected
Chlorobenzene	4300	State	Max Value = 4.6
Acetone	700	State	Max Value = 11.5
Methylene Chloride	5	MCL	Undetected
1,2 Dichloroethane	5	MCL	Undetected
Chloroform	100	NIPDWR	Undetected
Trichloroethane	3-200	MCLG,MCL	Undetected
1,1,2,2 Tetrachloroethane	none		Undetected
Toluene	2000	MCL	Max Value=92
Ethylbenzene	700	MCL	Max Value = 620
Trichlorofluoromethane	none		Undetected
Xylenes	1000	State	Undetected
Metals			
Arsenic	50	State	Undetected
Lead	5	MCL	Undetected
Mercury	2	MCL	Undetected
Zinc	5000	SMCL	Max Value = 11
Copper	1300	MCL	Undetected
Silver	50	NIPDWR	Undetected
Thallium	.400	MCLG	Undetected
Nickel	100	MCLG	Max Value = 230

1. Based on both water and fish consumption; fish consumption is the predominant source of exposure in deriving the WCQ. WQC reflectes a 10^{-5} lifetime risk level.

(2) Values for these compounds have been set the same as that for gamma-BHC (Lindane) for which there is an MCL.

TABLE 5-2
APPLICABLE STANDARDS AND GUIDELINES FOR AIR IN COMPARISON TO LEVELS AT THE PARCEL 18 SITE

Compounds	MASSACHUSETTS ALLOWABLE AMBIENT LIMIT (AAL) AIR ug/m^3	MASSACHUSETTS ALLOWABLE THRESHOLD CONCENTRATION (ATC) AIR ug/m^3	SITE CONDITIONS PARCEL 18 ug/m^3	NOTES
Carcinogenic PAH Compounds				
Benzo(a)pyrene	none	none	.0055	(1)
Benzo(a)anthracene	none	none	.0003	(1)
Benzo(b)fluoranthene	none	none	.0019	(1)
Benzo(k)fluoranthene	none	none	.0019	(1)
Benzo(g,h,i)perylene	none	none	.0003	(1)
Chrysene	none	none	.0099	(1)
Dibenzo(a,h)anthracene	none	none	.0003	(1)
Indeno(1,2,3-c,d)pyrene	none	none	.0003	(1)
Noncarcinogenic PAH Compounds				
Naphthalene	14.250	71	.0028	(1)
Fluorene	none	none	.0030	(1)
Anthracene	none	none	.0339	(1)
Phenanthrene	none	none	.0351	(1)
Fluoranthene	none	none	.0003	(1)
Pyrene	none	none	.0239	(1)
Acenaphthene	none	none	.0029	(1)
Pesticides				
Aldrin	none	none	.0002	(1)
Dieldrin	none	none	5.0000E-6	(1)
Heptachlor	.001	1	5.0000E-6	(1)
Heptachlor Epoxide	none	none	5.0000E-6	(1)
DDTR*	none	none	9.0000E-5	(1)
Endosulfan I	none	none	3.0000E-5	(1)
Endosulfan II	none	none		(1)
Endosulfan sulfate	none	none	1.0000E-5	(1)
Alpha-BHC	none	none	5.0000E-6	(1)
Beta-BHC	none	none	5.0000E-6	(1)
Gamma-BHC	.003	1	5.0000E-6	(1)
Methoxychlor	none	none		(1)
Total Arochlors	.0005	.02	.0001	(1)

TABLE 5-2
APPLICABLE STANDARDS AND GUIDELINES FOR AIR IN COMPARISON TO LEVELS AT THE PARCEL 18 SITE

Compounds	MASSACHUSETTS ALLOWABLE AMBIENT LIMIT (AAL) AIR ug/m^3	MASSACHUSETTS ALLOWABLE THRESHOLD CONCENTRATION (ATC) AIR ug/m^3	SITE CONDITIONS PARCEL 18 ug/m^3	NOTES
Volatile Organic Compounds				
Benzene	.120	9	< 6 ug/m^3	(2)
Chlorobenzene	6.260	470	NA	(2)
Acetone	160.540	800	NA	(2)
Methylene Chloride	9.500	47	NA	(2)
1,2 Dichloroethane	.040	55	NA	(2)
Chloroform	.040	660	NA	(2)
1-1-1 Trichloroethane	1038	5,200	Max = 1125 ug/m^3	(3)
1,1,2,2 Tetrachloroetha	.020	93	NA	(2)
Toluene	10.240	51	Max = 50 ug/m^3	(2)
Ethylbenzene	118.040	590	< 87 ug/m^3	(2)
Trichlorofluoromethane	none	none	NA	(2)
Xylenes	11.800	60	<22 ug/m^3	(2)
Metals				
Arsenic	none	none	.0085	(1)
Lead	.070	1	.1035	(1)
Mercury	none	none	.0004	(1)
Zinc	none	none	.1062	(1)
Copper	.540	3	.0225	(1)
Silver	none	none	.0012	(1)
Thallium	none	none	.0087	(1)

1. Estimated assuming that surface soils become suspended in site air at a concentration of 90 ug/m^3 as PM-10. The concentration shown is the maximum calculated for an exposure scenario for Phases I, II, or III.
2. Concentration measured in soil gas.
3. Concentration of 1-1-1 trichloroethane was estimated from chromatograph. The compound was suspected of being present in three of twenty-three samples.

TABLE 5-3 TOTAL SITE RISKS

Phase I Risks

A. Risk To Construction Workers	Hazard Index	Carcinogenic Risk
Foundation Construction	0.038	2.42E-07
Building Construction	0.156	2.73E-06
Total Phase I Worker Risk	0.194	2.97E-06
B. Risk To Pedestrian Commuters	0.0005	7.52E-11
C. Risk To Off-Site Children	0.003	4.56E-10

Phase II Risks

A. Risk To Construction Workers	Hazard Index	Carcinogenic Risk
Foundation Construction	0.029	3.21E-07
Building Construction	0.153	7.56E-07
Total Phase II Worker Risk	0.182	1.08E-06
B. Risk To Pedestrian Commuters	0.0006	5.47E-11
C. Risk To Off-Site Children	0.006	5.11E-10

Phase III Risks

A. Risk To Construction Workers	Hazard Index	Carcinogenic Risk
Foundation Construction	0.033	3.03E-07
Building Construction	0.153	1.51E-06
Total Phase III Worker Risk	0.186	1.81E-06
B. Risk To Pedestrian Commuters	0.0005	1.09E-10
C. Risk To Off-Site Children	0.006	1.03E-09

TABLE 5-3. TOTAL SITE RISKS (CONT.)

Post Construction Risks

	Hazard Index	Carcinogenic Risk
A. Risk To Daycare Children	0.18	2.02E-06
B. Risk To Emergency Worker	Risks Estimated To Be Less Than Risk To Construction Workers	
C. Risk To Maintenance Workers	Risks Estimated To Be Less Than Risk To Construction Workers	
D. Risk To Office Workers	Minimal Exposure Based Upon Foreseeable Site Use And Soil Gas Data	

Current Risks

A. Incidental Trespassers	Risks Estimated To Be Less Than Risk To Construction Workers

Figures

FIGURE 2-2 COMPARISON OF ARSENIC SOIL LEVELS TO SELECTED BACKGROUND

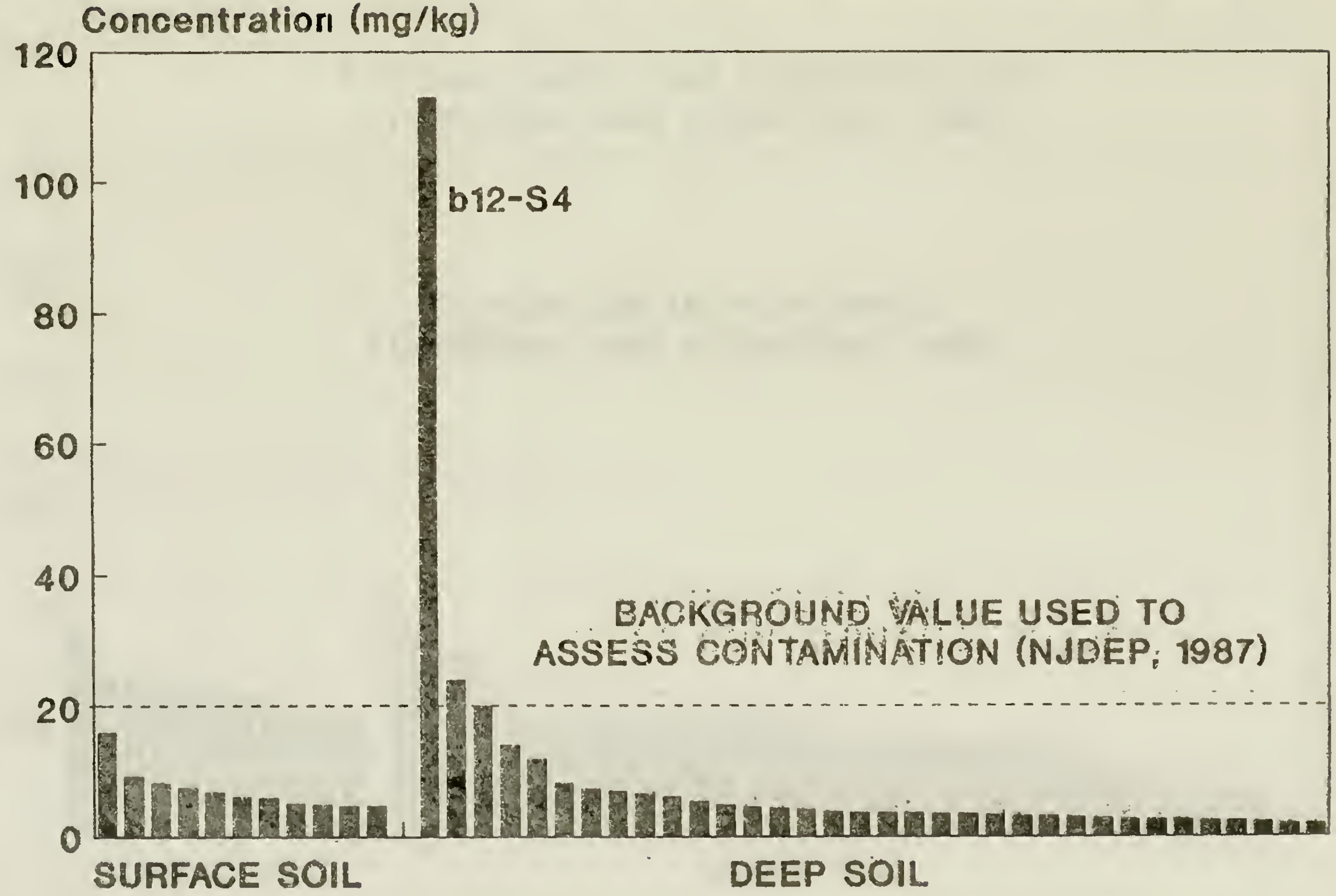


FIGURE 2-3 COMPARISON OF BERYLLIUM SOIL LEVELS TO SELECTED BACKGROUND

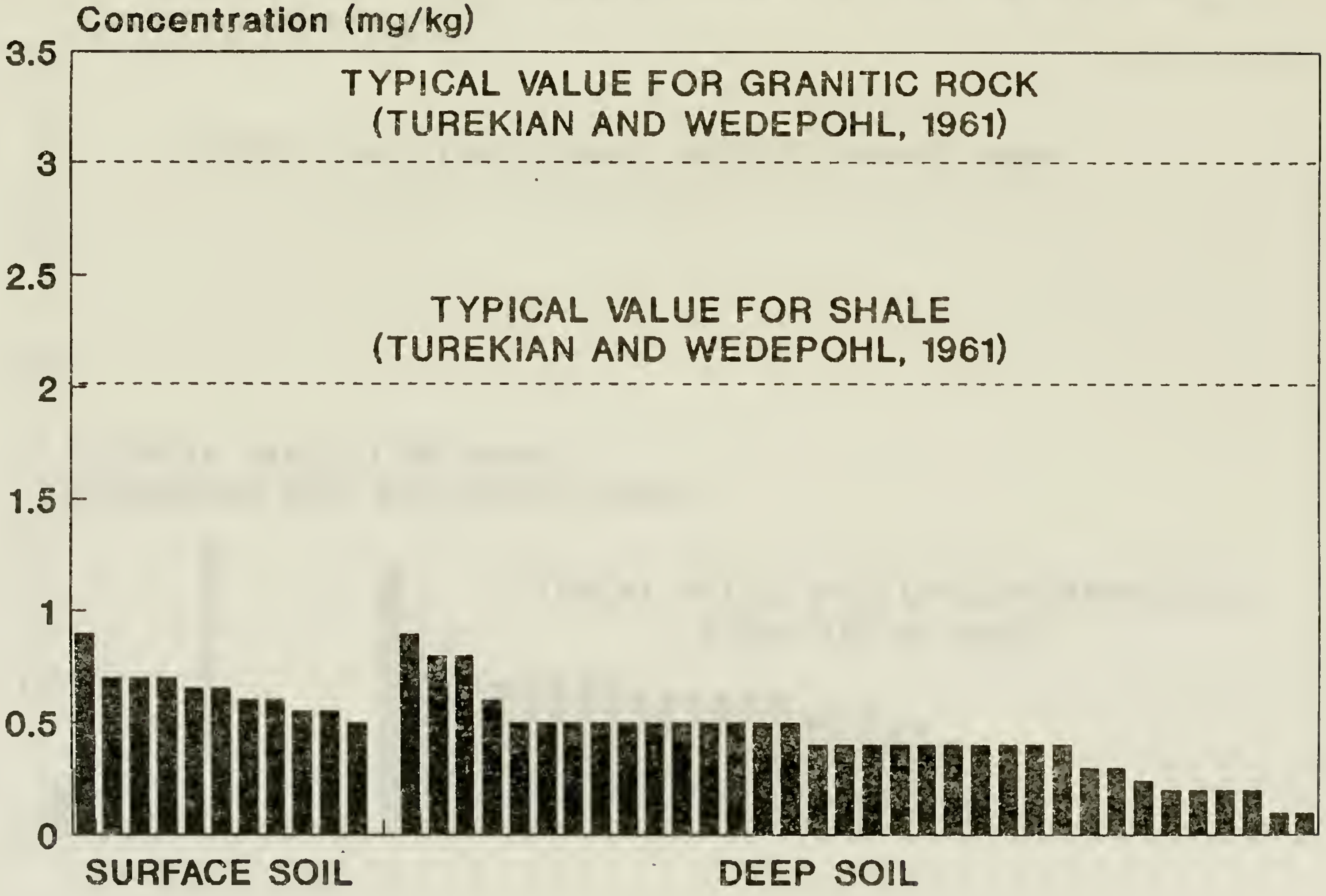


FIGURE 2-4 COMPARISON OF CADMIUM SOIL LEVELS TO SELECTED BACKGROUND

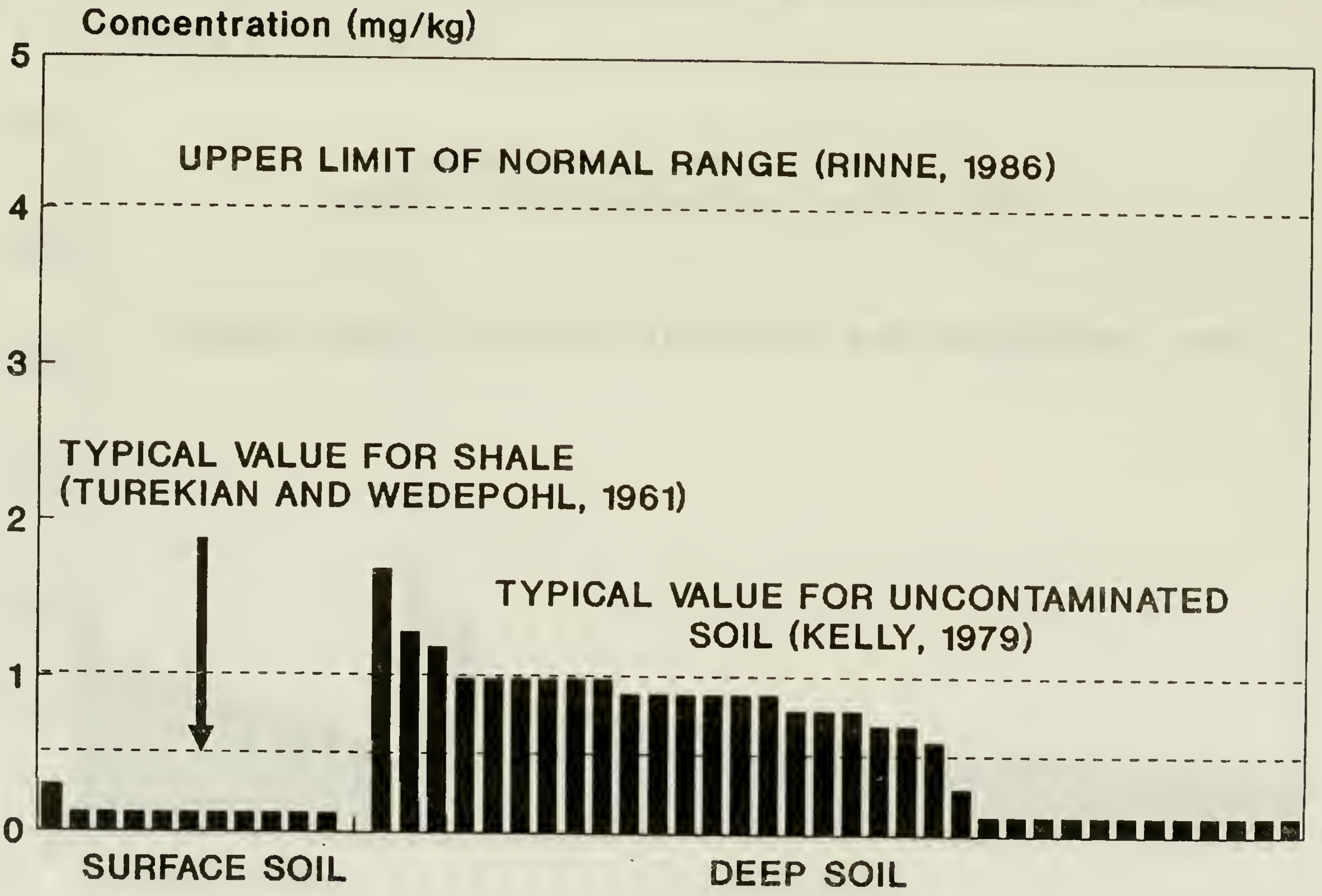


FIGURE 2-5 COMPARISON OF CHROMIUM SOIL LEVELS TO SELECTED BACKGROUND

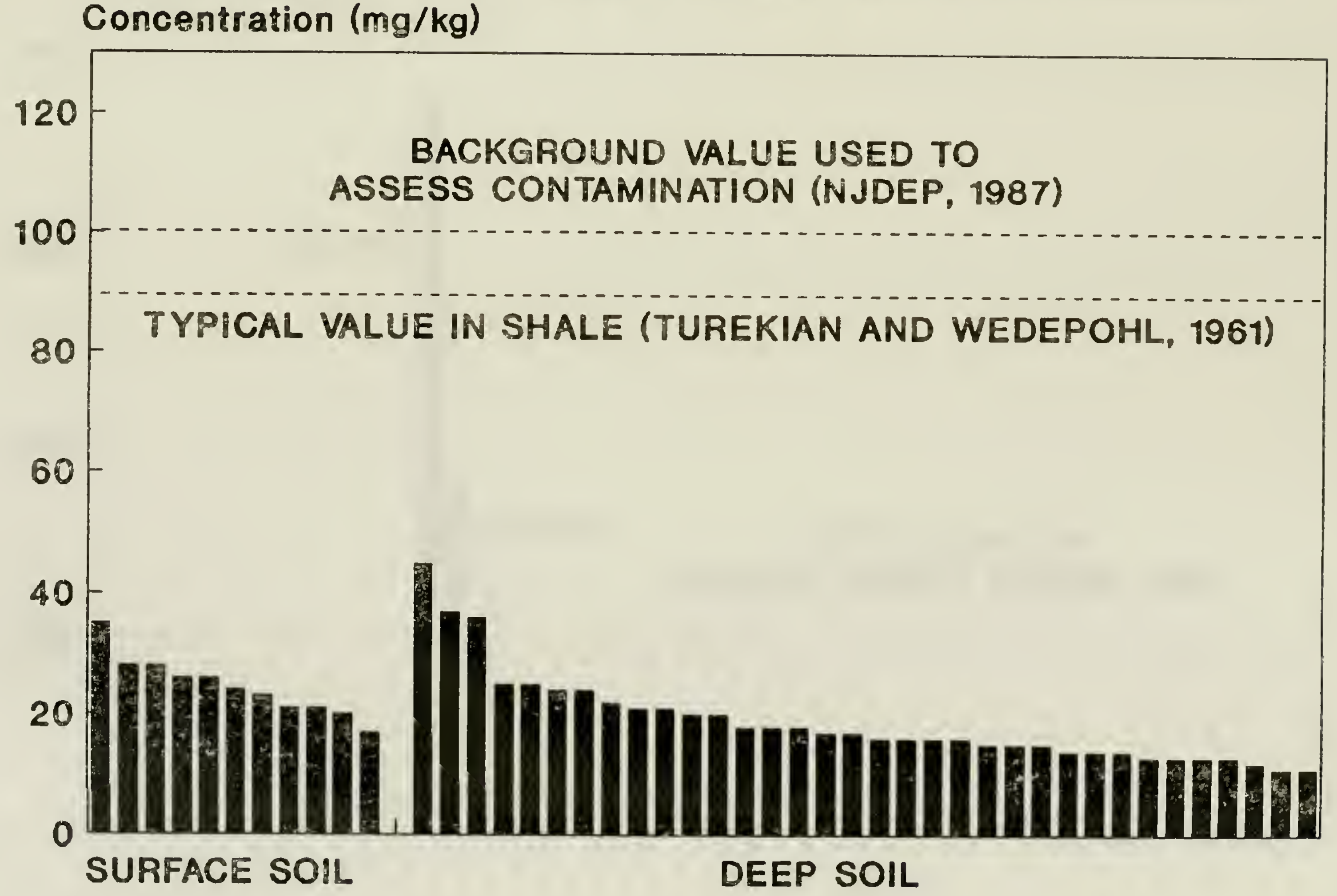


FIGURE 2-6 COMPARISON OF COPPER SOIL LEVELS TO SELECTED BACKGROUND

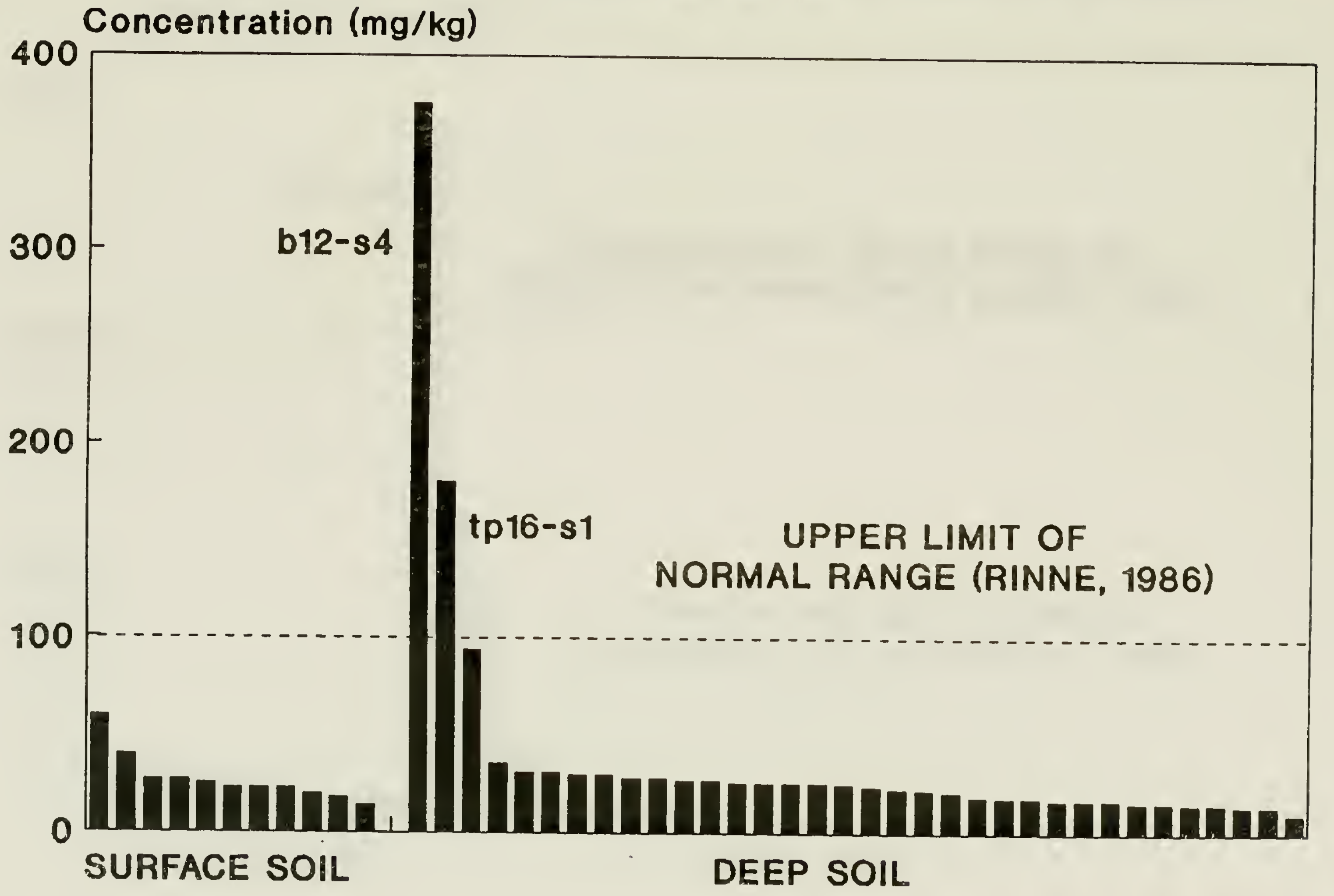


FIGURE 2-7 COMPARISON OF LEAD SOIL LEVELS TO SELECTED BACKGROUND

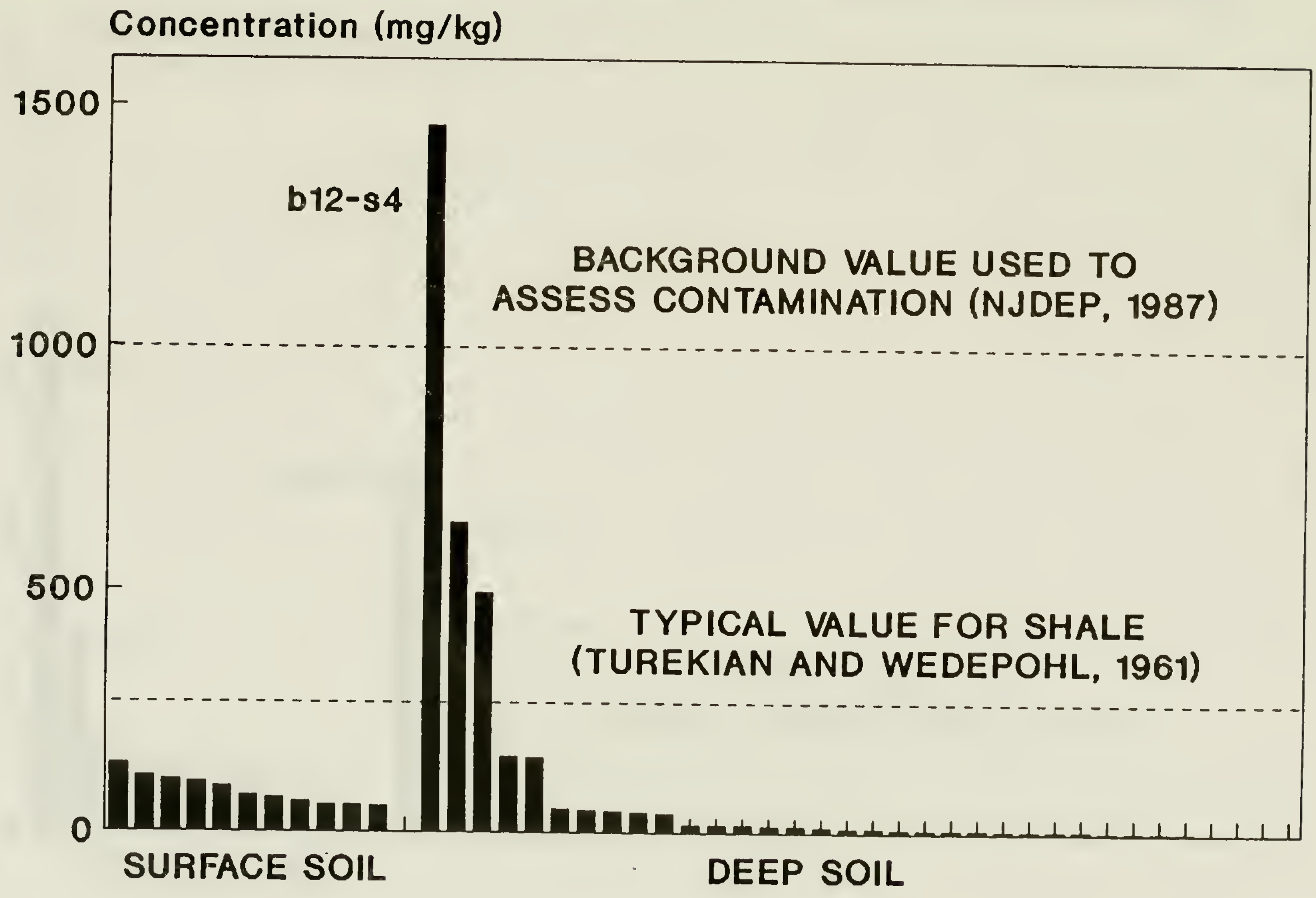


FIGURE 2-8 COMPARISON OF MERCURY SOIL LEVELS TO SELECTED BACKGROUND

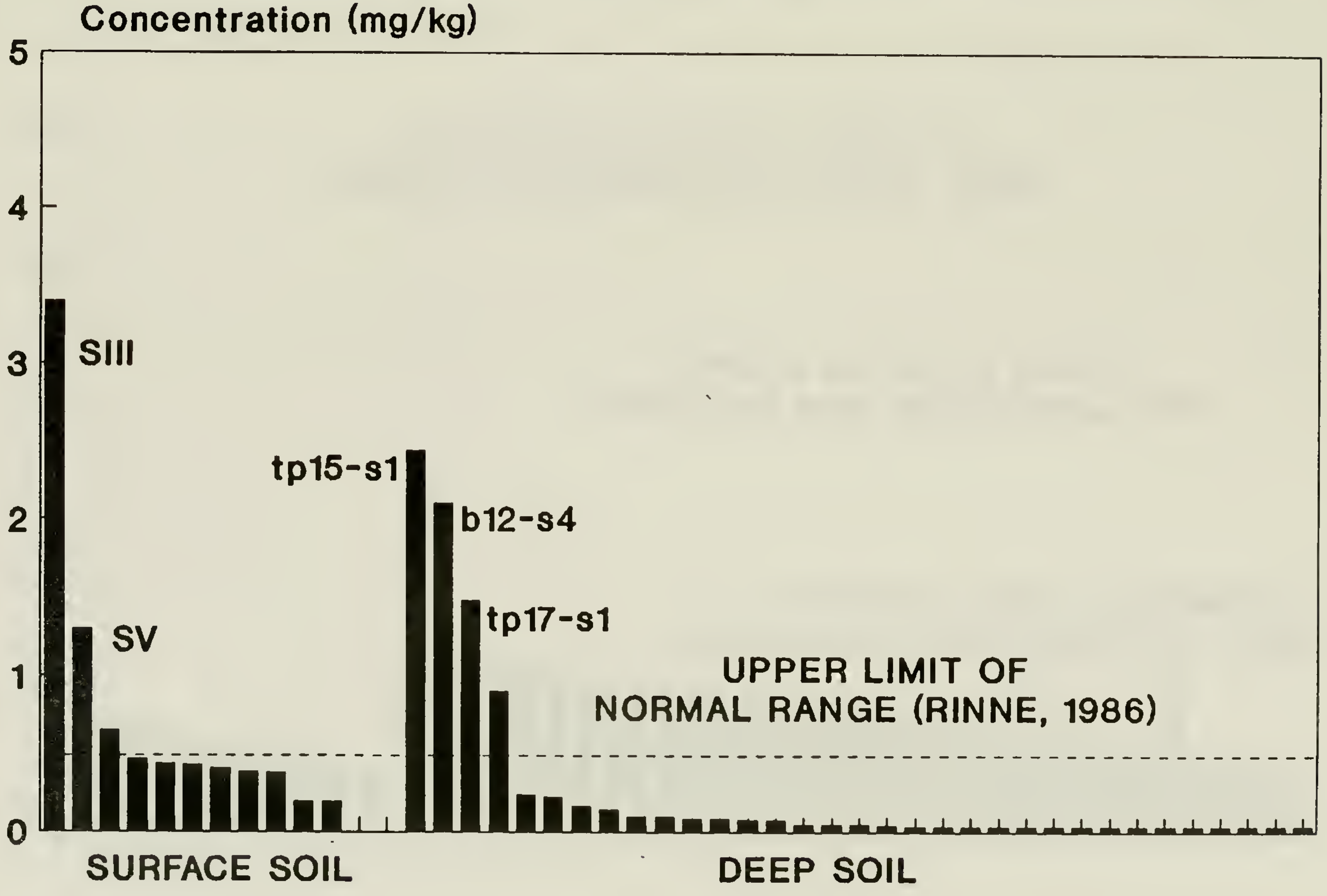


FIGURE 2-9 COMPARISON OF NICKEL SOIL LEVELS TO SELECTED BACKGROUND

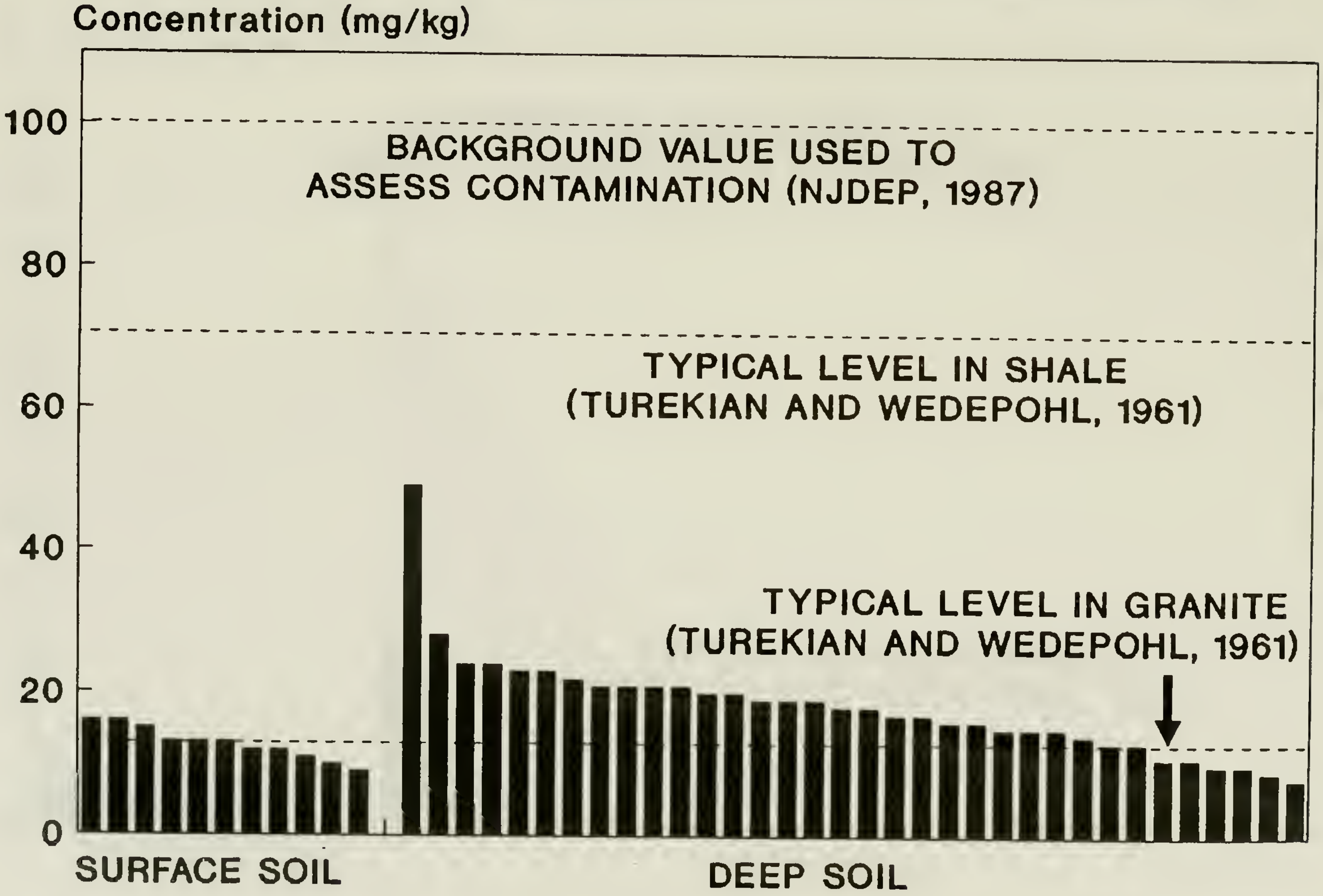


FIGURE 2-10 COMPARISON OF SELENIUM SOIL LEVELS TO SELECTED BACKGROUND

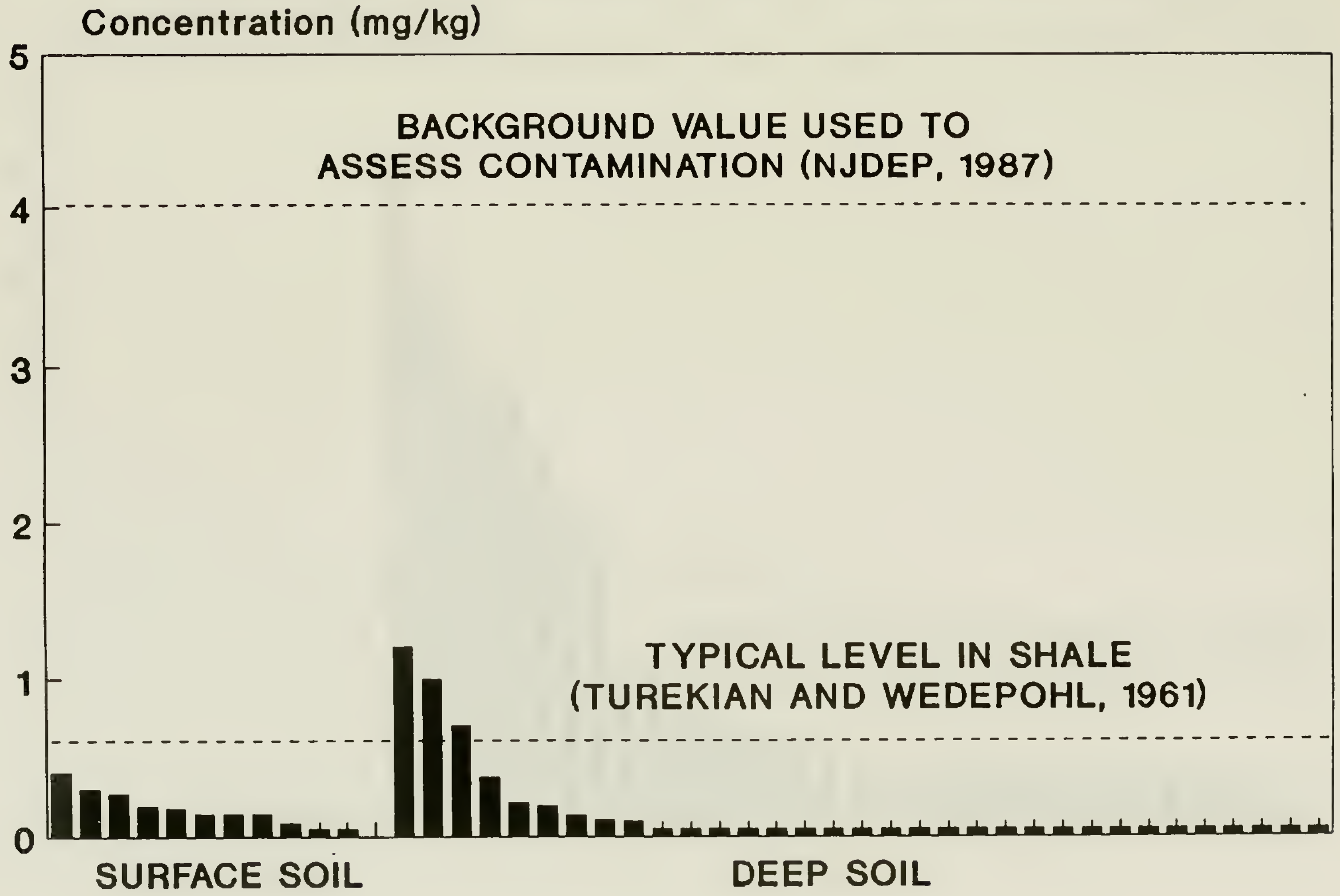


FIGURE 2-11 COMPARISON OF SILVER SOIL LEVELS TO SELECTED BACKGROUND

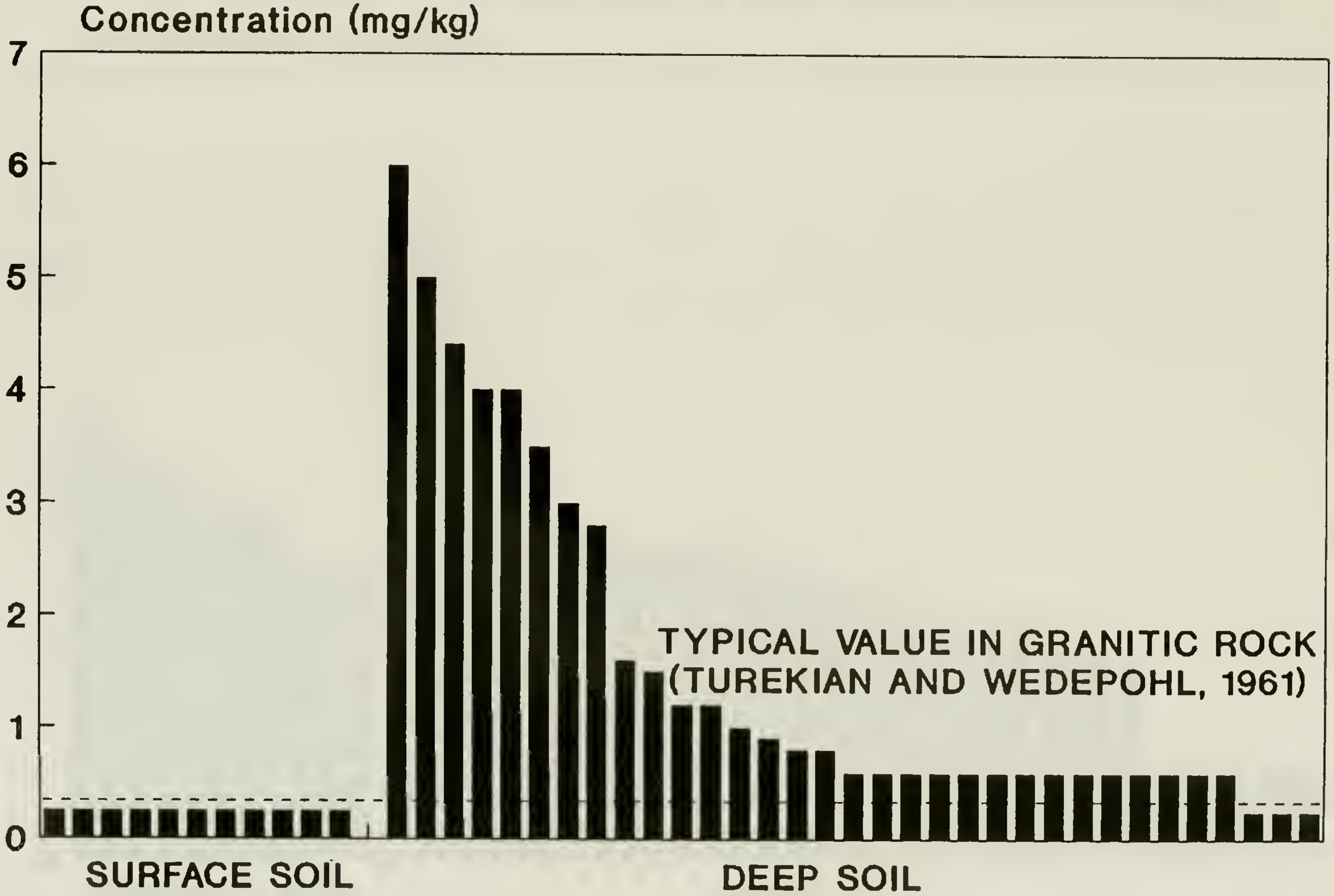


FIGURE 2-12 COMPARISON OF THALLIUM SOIL LEVELS TO SELECTED BACKGROUND

where is background?

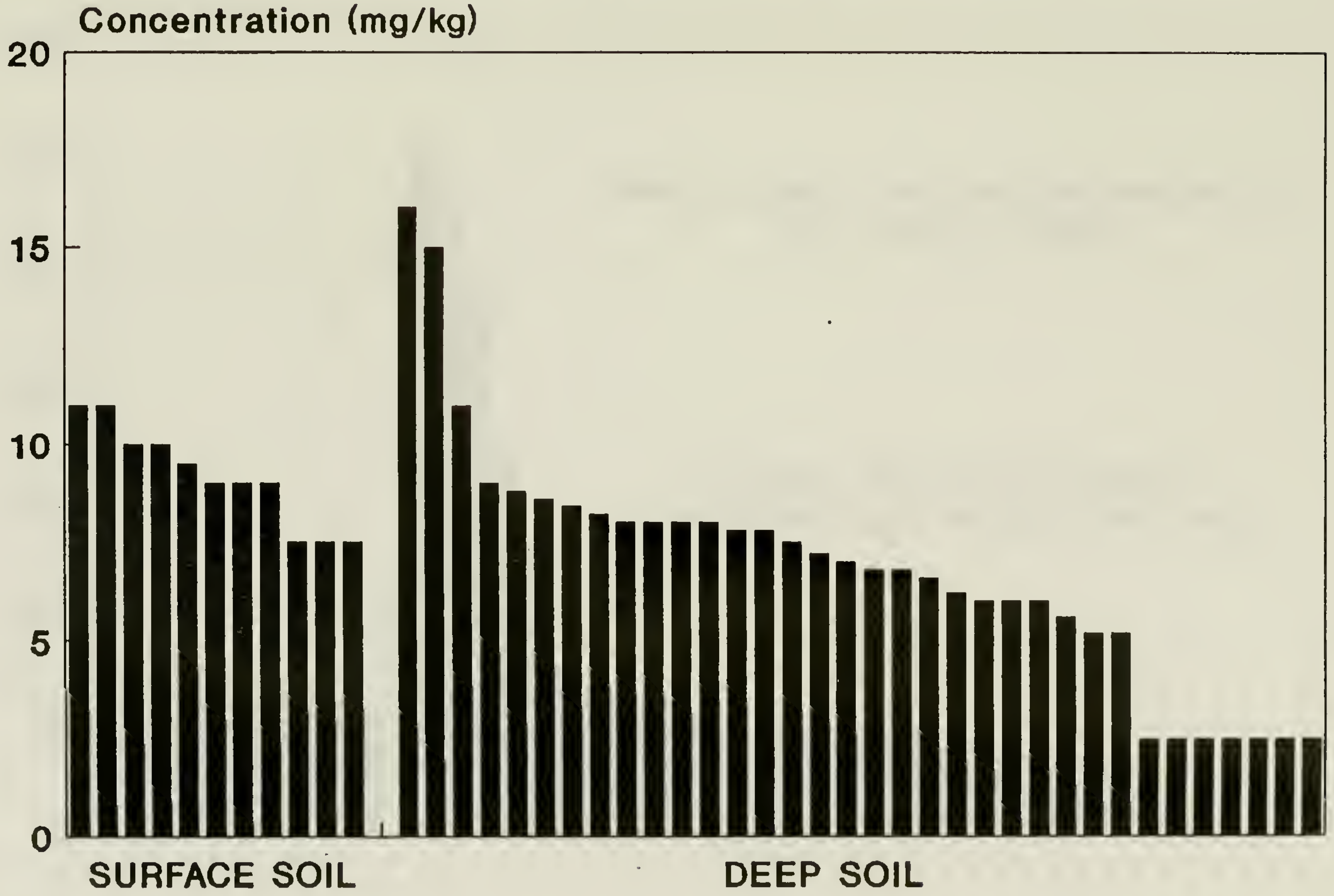


FIGURE 2-13 COMPARISON OF ZINC SOIL LEVELS TO SELECTED BACKGROUND

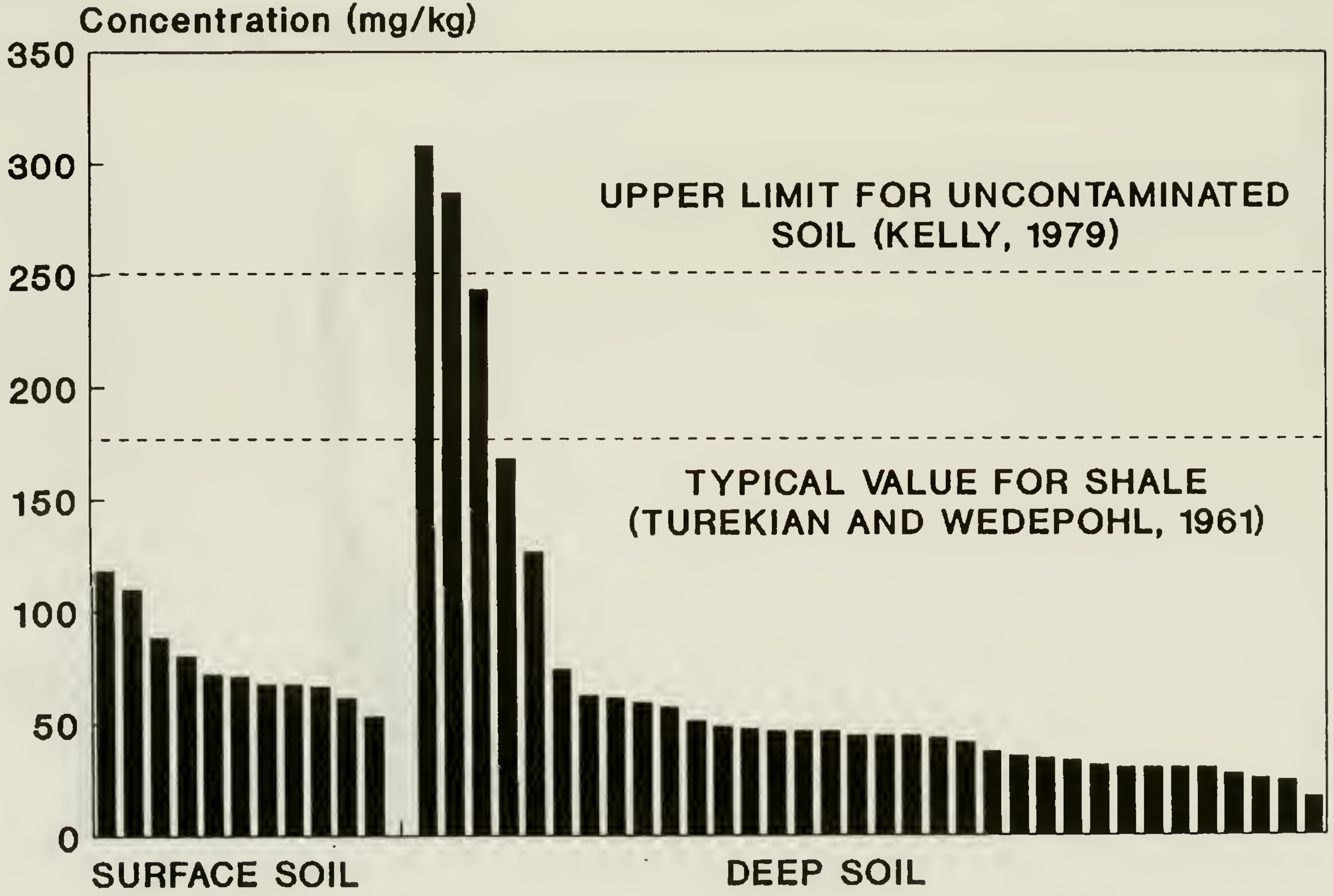


FIGURE 2-14 TOTAL PESTICIDES IN SURFACE AND DEEP SOIL

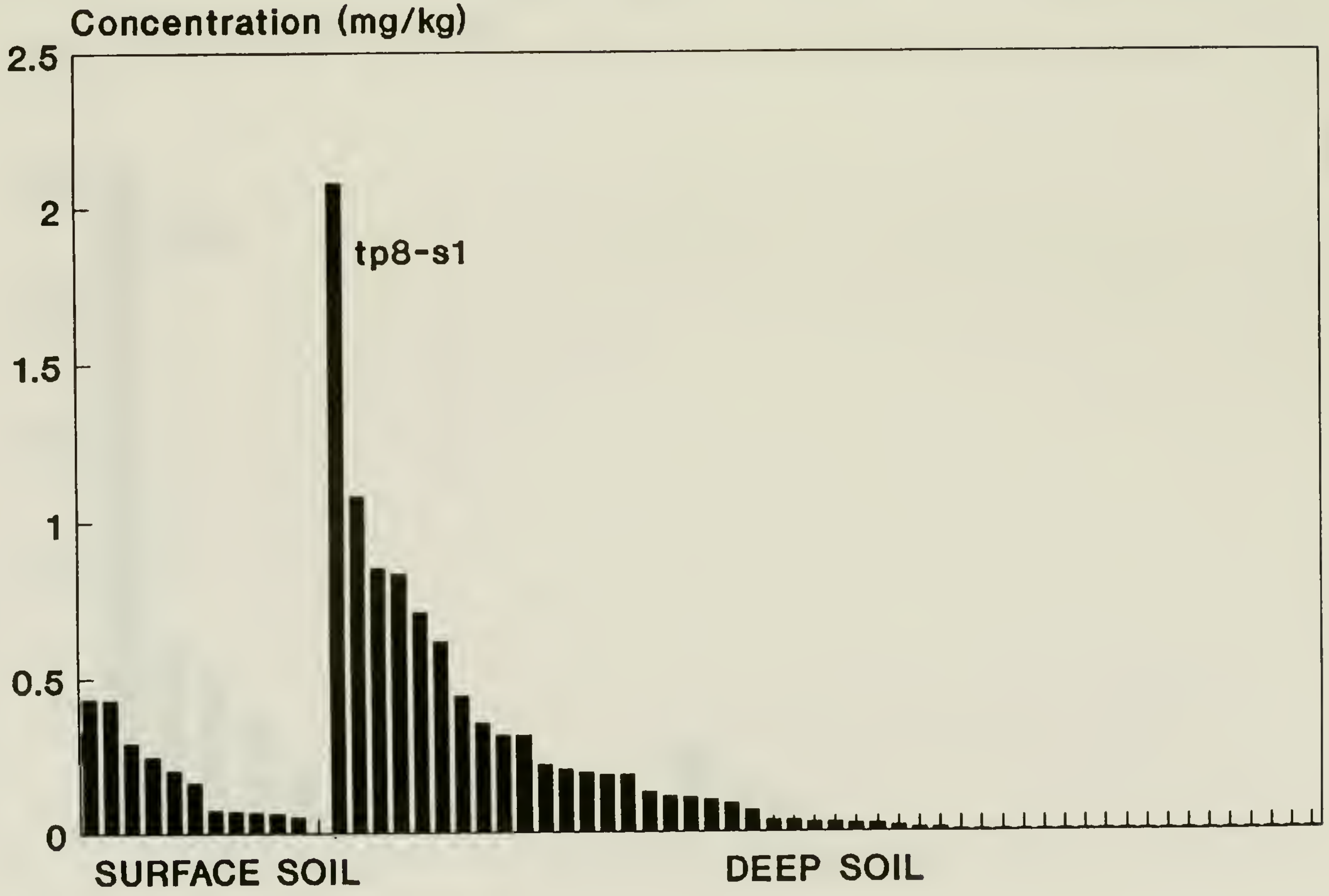


FIGURE 2-15 TOTAL BASE/NEUTRALS IN SURFACE AND DEEP SOIL

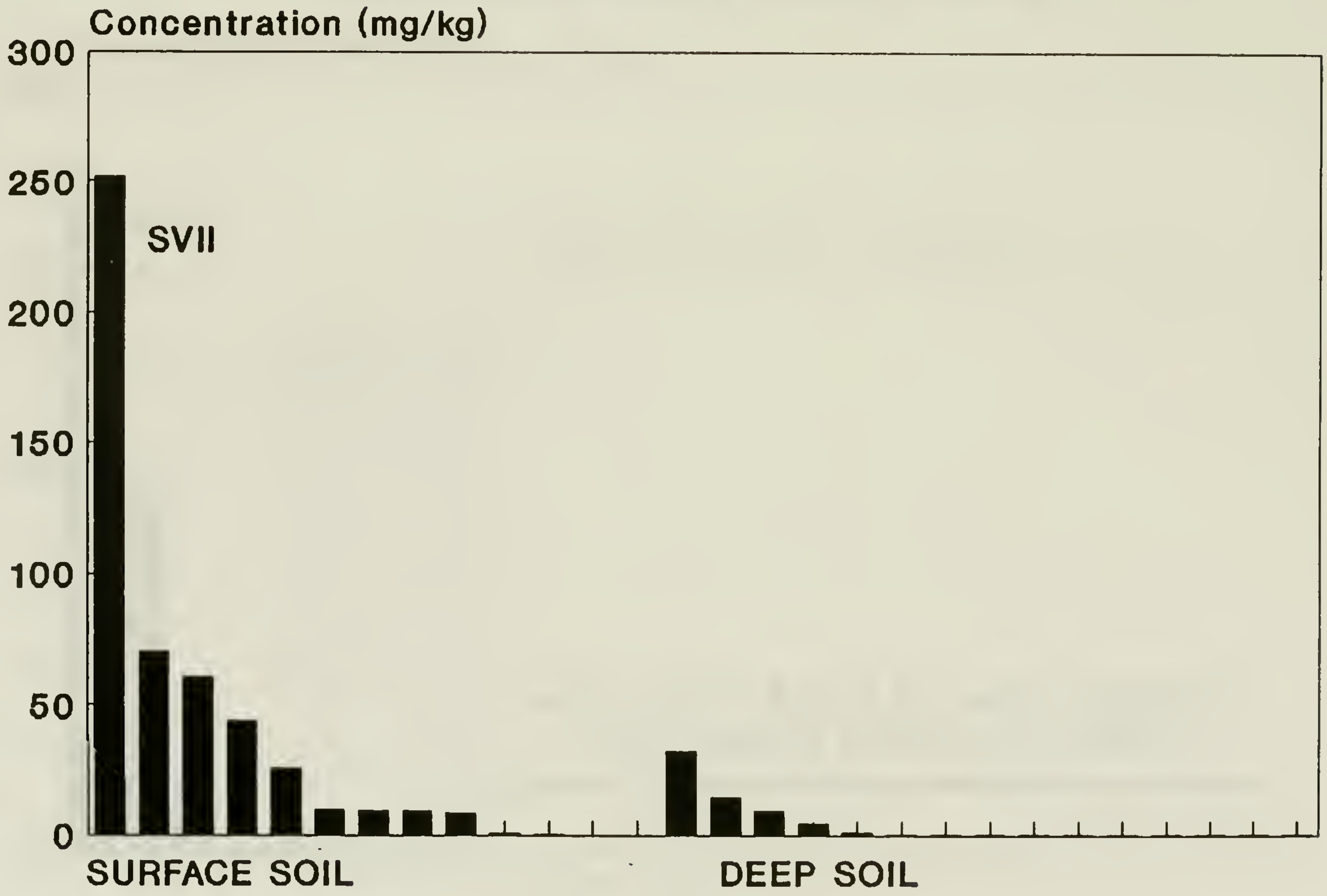


FIGURE 2-16 TOTAL VOCS IN SURFACE AND DEEP SOIL

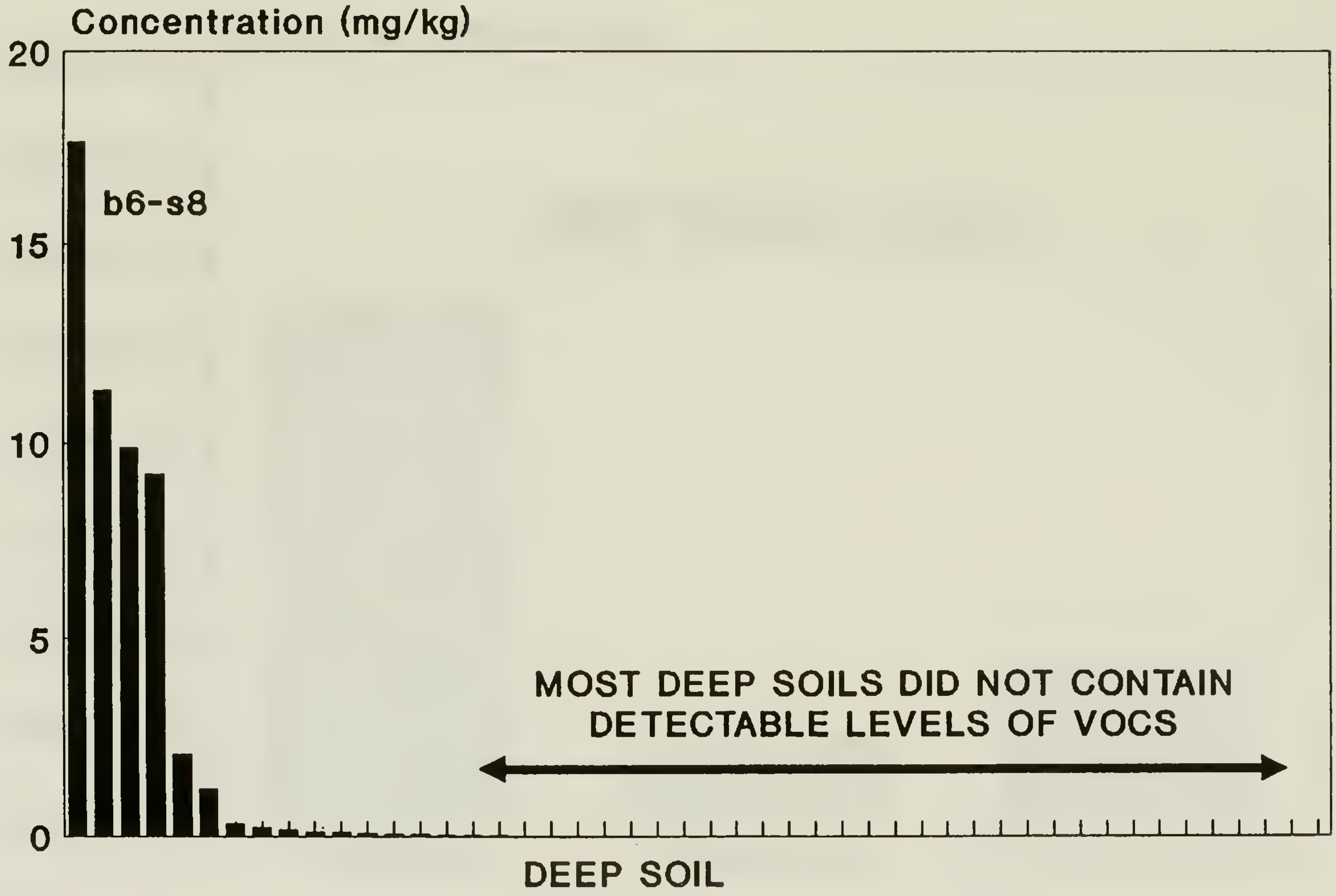


FIGURE 5-1 CARCINOGENIC RISKS
DURING PHASE I CONSTRUCTION

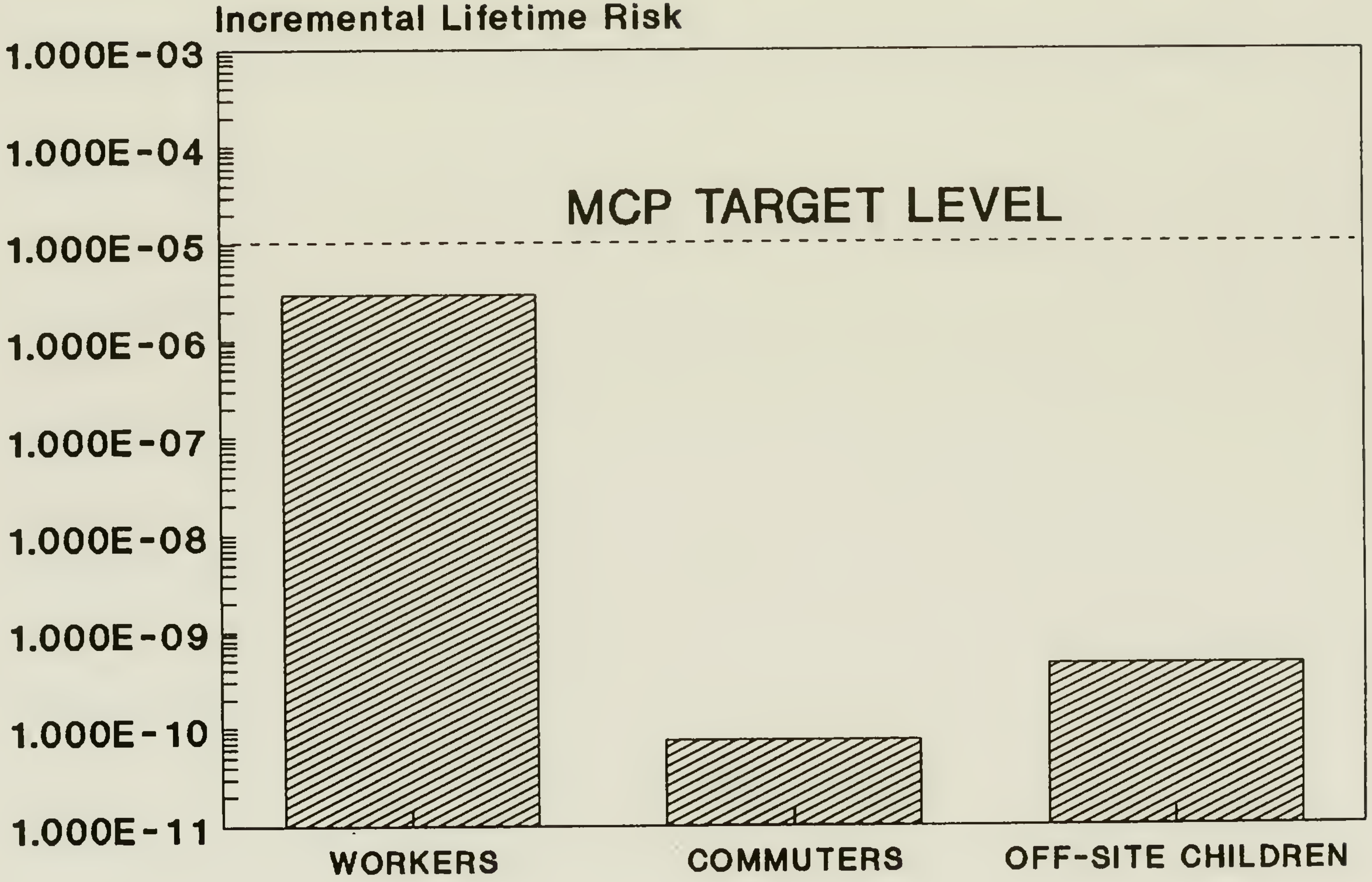


FIGURE 5-2 CARCINOGENIC RISKS
DURING PHASE II CONSTRUCTION

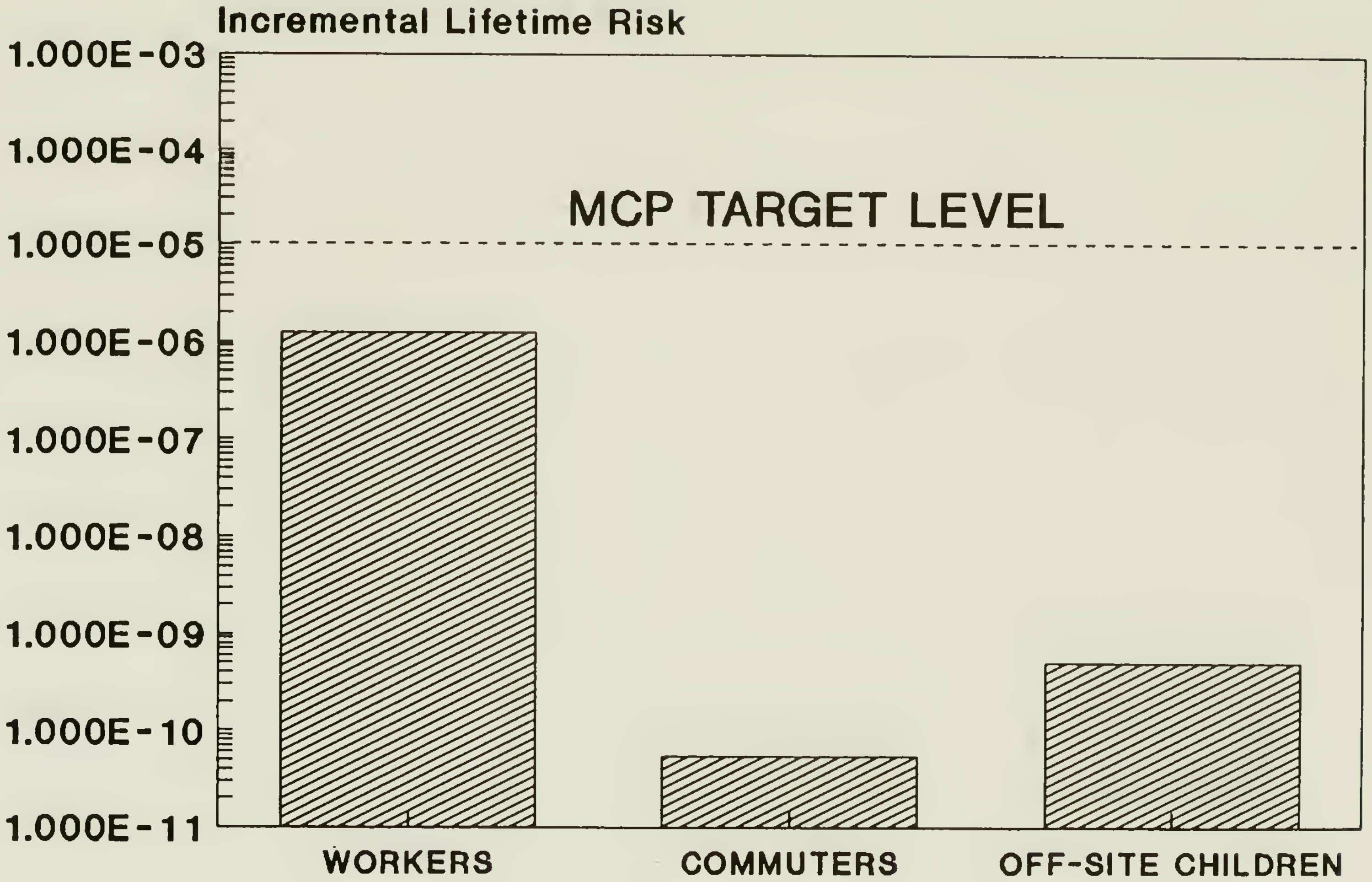


FIGURE 5-3 CARCINOGENIC RISKS
DURING PHASE III CONSTRUCTION

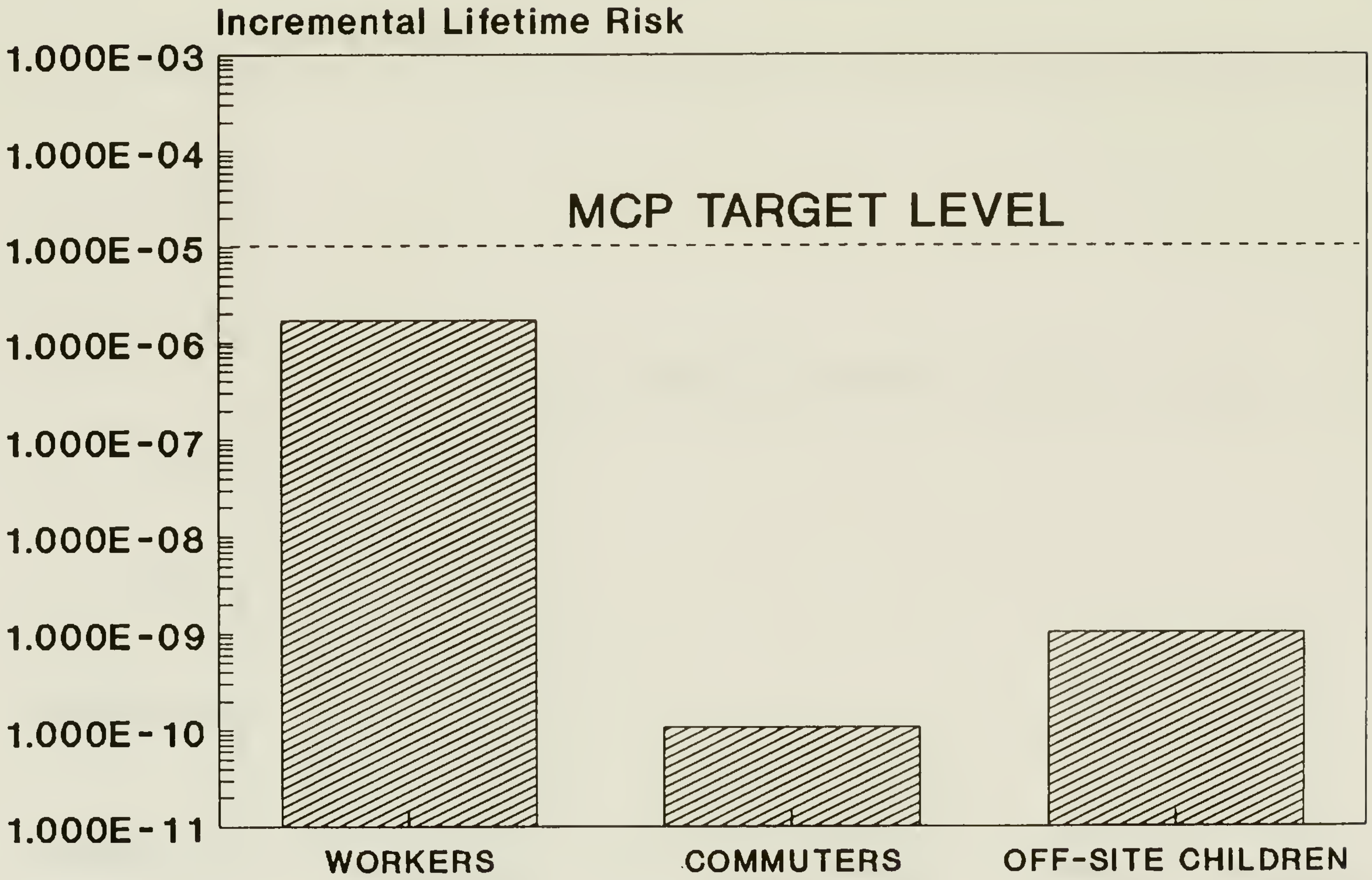


FIGURE 5-4 HAZARD INDEX RATIOS
DURING PHASE I CONSTRUCTION

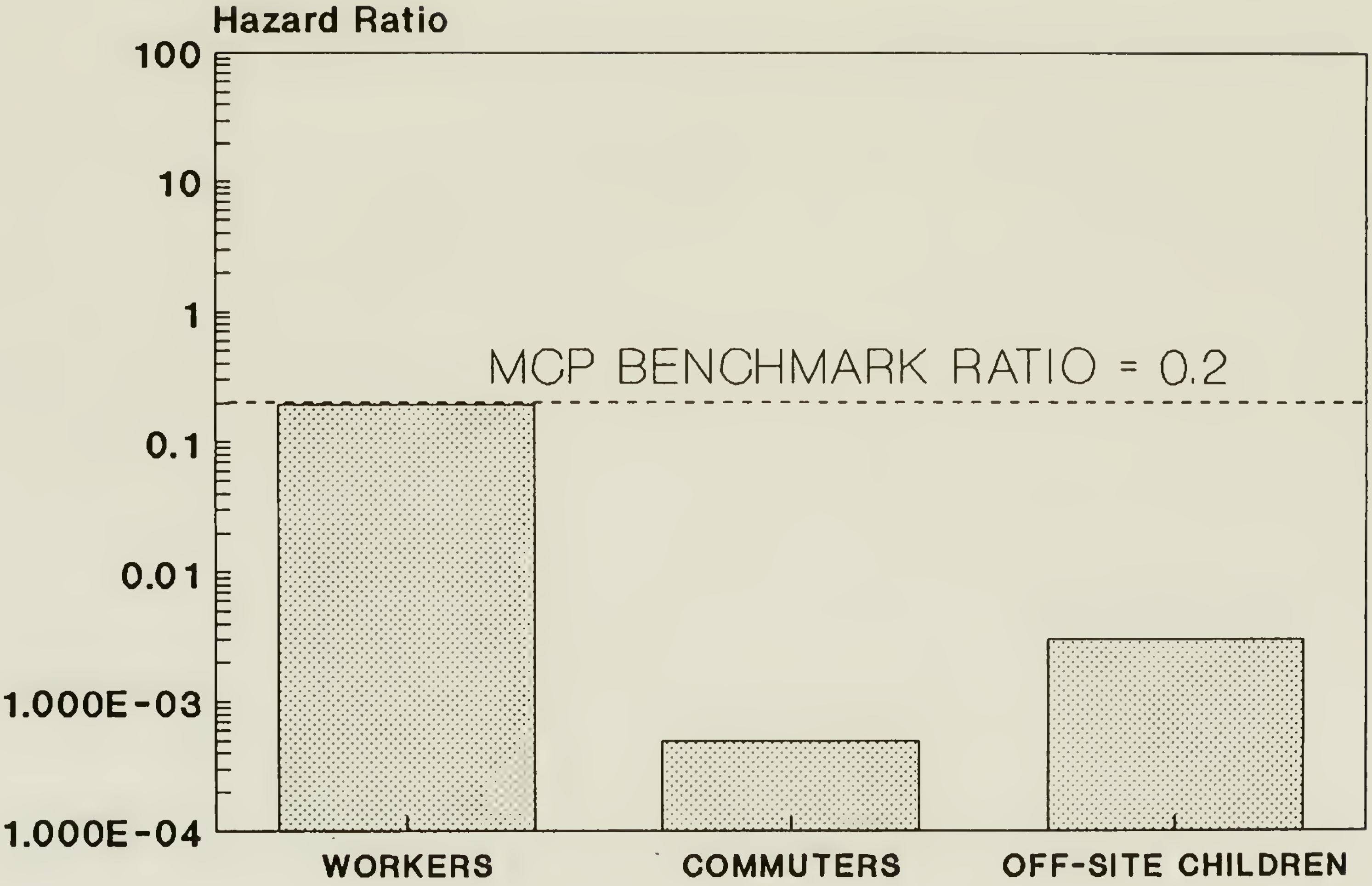


FIGURE 5-5 HAZARD INDEX RATIOS
DURING PHASE II CONSTRUCTION

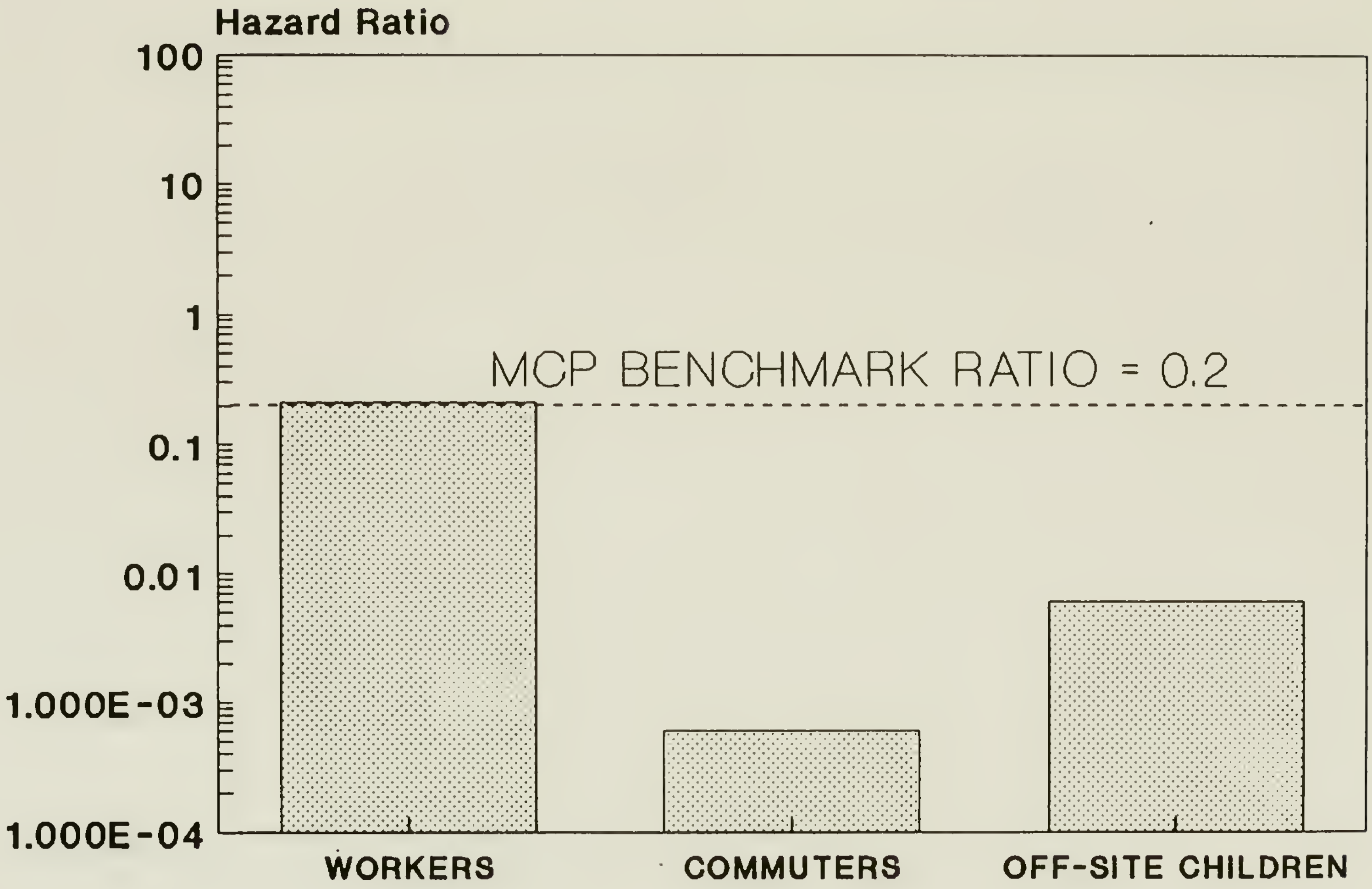
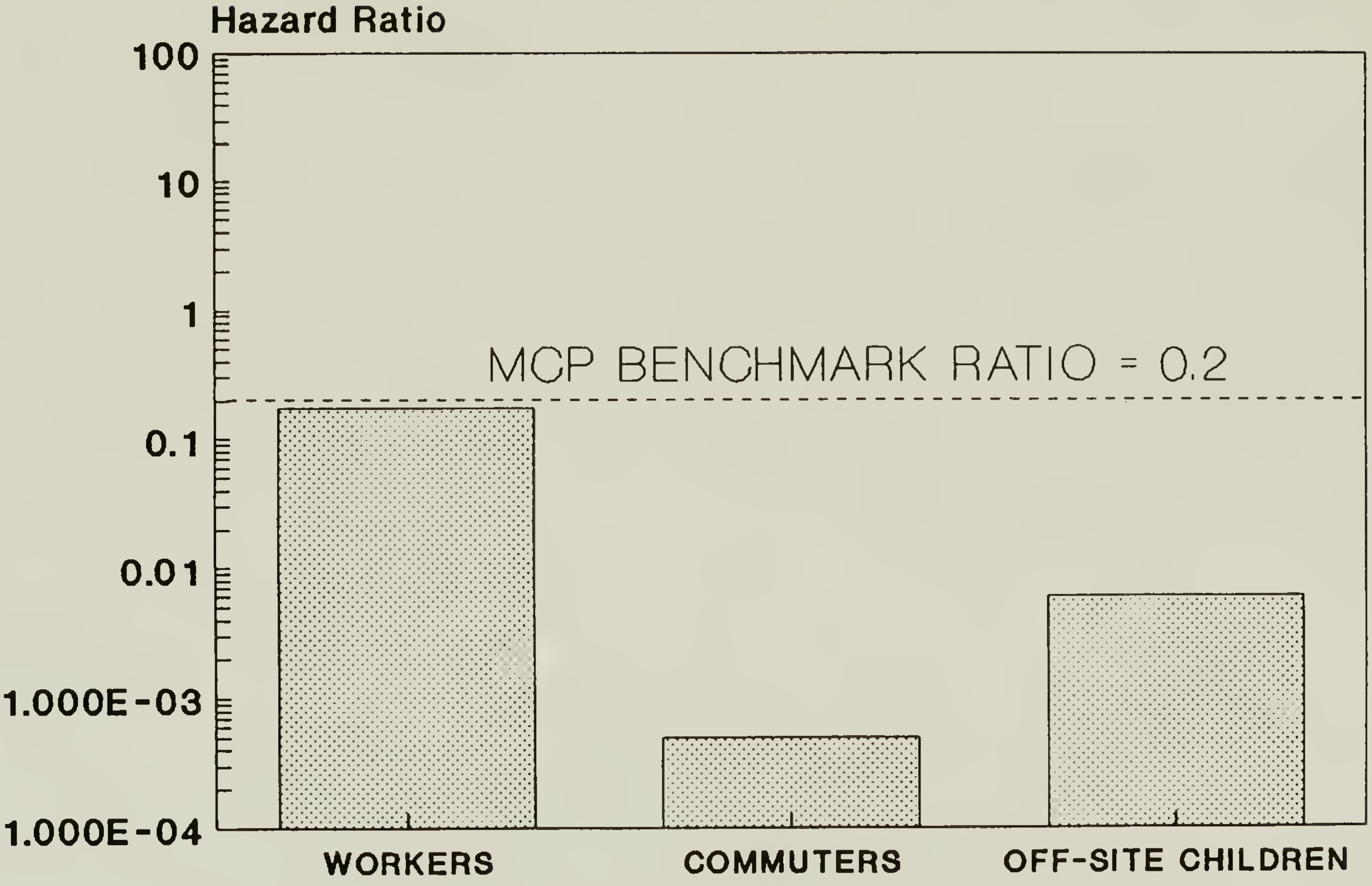


FIGURE 5-6 HAZARD INDEX RATIOS DURING PHASE III CONSTRUCTION



Appendix A

APPENDIX A
CHEMICAL PROFILES

BENZO(a)PYRENE

Information presented below has been assembled to aid in the preparation of risk assessments. Detailed data on health end environmental effects can be found in the bibliography provided at the end of this profile.

A. Physical and Chemical Properties

Property (a)	Value	Reference
Molecular weight	252.32 g/mol	Verschueren 1983
Water solubility	3.80 x 10-3 ppm at 25oC	Mabey et al. 1982
Vapor pressure	5.60 x 10-9 mm Hg at 25oC	Mabey et al. 1982
Koc	5.50 x 10E6	Mabey et al. 1982
Kow	1.15 x 10E6	Mabey et al. 1982
Kh	negligible	Mabey et al. 1982
Hc	4.90 x 10-7 atm-m3/mol	Mabey et al. 1982

aKoc = Organic carbon-water partition coefficient Kow = Octanol-water partition coefficient; Kh - Hydrolysis rate constant; Hc = Henry's Law constant.

B. Toxic Effects to Humans

Exposure to BaP has been associated with lung, skin, and other tumors in man. But because BaP exposure always occurs in the presence of other PAHs, it has not been possible to determine definitely that BaP is the causative agent in the production of these tumors (IARC 1983).

USEPA's Carcinogen Assessment Group estimated unit cancer risks (UCRs) for BaP of 11.5 and 6.1 (mg/kg/day)-1 by the oral and inhalation routes of exposure (USEPA 1984).

ICF-Clement (1987) has utilized a biologically-based model to estimate the carcinogenic potency of BaP. The model yields point estimates of 5.74 (mg/kg/day)-1 for exposure via the oral route and 0.4533 (mg/kg/day)-1 for exposure via the inhalation route.

Data used to estimate carcinogenic potency of BaP via the oral route were taken from a series of studies conducted by Neal and Rigdon (1967). A number of experimental factors that are atypical of the standard bioassay protocol were employed in the Neal and Rigdon (1967) study. These included variable age of

first exposure, a duration of exposure that lasted about one-seventh of a lifetime, and an observation period that was less than one-fifth of a lifetime. USEPA (1982) pointed out several qualifications relating to the use of the data of Neal and Rigdon. First, rodent forestomach is different anatomically from human stomach - the mouse cells being similar to human epithelial cells. Thus, they may represent an inappropriate assay of BaP carcinogenesis by the oral route due to the potency of BaP as a skin carcinogen. Second, the mice were exposed to BaP for a period of time considerably shorter than their lifetimes. And third, qualitative and quantitative differences exist in the metabolism of BaP between rodents and man. Data used to estimate carcinogenic potency via the inhalation rate were taken from Thyssen et al. (1981).

C. Pharmacokinetics

There are no pharmacokinetic data for BaP in humans (USEPA 1980). Animal data indicate that BaP is readily absorbed after exposure by inhalation or oral intake and distributes to many tissues in the body (USEPA 1980). BaP itself is not believed to be carcinogenic but requires metabolic activation before carcinogenesis can be expressed. BaP is metabolized primarily by the hepatic cytochrome P-450 dependent mixed function oxidase system (often referred to as the aryl hydrocarbon hydrolase, AHH). Aryl hydrocarbon hydroxylase is readily inducible by exposure to a variety of chemicals, including BaP, and is found in most adult as well as fetal mammalian tissues. It catalyzes the formation of reactive epoxide intermediates which are capable of forming covalent bonds with cellular macromolecules such as DNA, RNA, and proteins and ultimately may result in tumor formation. The currently suspected ultimate carcinogenic derivative of BaP is BaP-7,8-diol-9,10-epoxide (USEPA 1982).

D. Environmental Effects

Because there is very limited information concerning the environmental effects of specific PAH compounds, a general overview is presented here. Obviously some coumpounds will be more toxic than others. Infomation that is developed for specific compounds will be incorporated within specific chemical profiles. This section of the B[a]P chemical profile will be referenced within other PAH chemical profiles and is intended to be more generic in nature.

General Discussion Concerning Environmental Fate and Effects of PAH compounds: This class of contaminants includes the largest number of chemicals of interest identified at town gas sites. Therefore, in addition to providing information on specific compounds, where available, information is also provided on PAHs as a class.

No standard freshwater toxicity tests have been reported for any polynuclear aromatic hydrocarbon (above Naphthalene) as a class or specific compounds. There are some data for bioconcentration during tests with model ecosystems or for short periods of time.

Lu, et al. (1977) conducted studies with benzo[a]pyrene in a terrestrial-aquatic model ecosystem and observed bioconcentration factors after 3 days ranging from 930 for the mosquitofish to 134,248 for _Daphnia_ _pulex_ . Bioconcentration factors for _Daphnia_ _magna_ and _Hexagenia_ sp. for a shorter time were 200 to 3,500.

Some PAH metabolites are carcinogenic, mutagenic, or teratogenic. Rather than enhancing detoxification, metabolism of some carcinogenic PAHs in induced animals could result in a higher steady-state level of toxic products (Stegeman, 1981). Although studies with various carcinogens have demonstrated that chemicals can cause cancer in aquatic species, most attempts to demonstrate carcinogenesis by PAHs in aquatic species have produced equivocal results (Pliss and Khudoley, 1975). Although recently there has been some evidence that PAH can cause cancer in in aquatic animals, there is to date no demonstration of PAH-induced carcinogenisis in vertebrate or invertebrate species (Neff, 1979; Stegeman, 1981).

Recent studies in the Duwamish River, Boston Harbor, and Hudson River have identified populations of Dover sole and Atlantic tomcod with very high incidences of hepatocellular carcinoma (McCain et al., 1977; Smith et al., 1979), and higher incidences of similar diseases have been reported for other environments. Although the etiology of such diseases in fish is uncertain, there is reason to suspect that the chemical environment is resonsible, and PAHs have not been exonerated (Stegeman, 1981). The question of whether PAHs induce cancer in aquatic species, particulary at known ambient levels, is yet to be answered.

The impacts of concern in the terrestrial environment include both direct toxicity and food-chain impacts. The toxic effects of PAH in mammals can be inferred from the extensive toxicity testing work performed on laboratory animals. As with humans, the basic conclusion is that PAHs are only slightly to moderately toxic by acute exposure, but chronic exposures to certain PAHs can result in cancer. Biomagnification in animal food chains is unlikely, however, since PAHs are readily metabolized.

Plant Uptake

PAH compounds are widespread in soils as well as in crops, plants, and algae in the natural environment. Natural background levels of PAH are, in general, greatest in leafy materials of plants. Sims and Overcash (1983) reported that

tree leaves contained 22 to 88 parts per billion (ppb) and cereal grains 48 to 66 ppb PAH. The source of PAH in plants is subject to much debate. Elevated benzo(a)pyrene (BaP) has been found in soils and in above-ground vegetation adjacent to urban areas (Kolar et al. 1975 and Smirnov 1970 as cited in Sims and Overcash 1983). However, other researchers have shown that levels of PAH are independent of area of origin (Hancock et al. 1970, Graf and Diehl 1976, and Borneff et al. 1968, as cited in Sims and Overcash, 1983).

According to Sims and Overcash (1983), the presence of PAH in plants is a result of airborne deposition, biochemical synthesis, and plant-uptake from soil. Much of the plant-uptake research has been conducted with BaP and is not necessarily representative of other PAH (Sims and Overcash 1983). In studies with BaP, the ratio of crop/soil (c/s) residues of BaP was higher in stems and straw when compared to the seed concentration. This indicated biomagnification of BaP but not as a direct function of soil concentration. While biomagnification of BaP and 3,4 benzofluoranthene is evident, other constituents of PAH may not be taken up by plants. It may be that biomagnification of PAH is not only compound specific but also plant specific. The fate and impacts of PAH in terrestrial plant systems have recently been reviewed by Santodonato et al. (1981). These reviews conclude that the principal mechanism of PAH uptake by plants is by adsorption rather then absorption. For example, Harms (1975) showed that less the 0.2 percent of C14 BaP found in the roots of wheat was translocated to the shoots, and Blum and Swarbrich (1975) found no appreciable translocation of C14 BaP from the roots in green beans, cantaloupes, and cotton. In other words, airborne PAH will accumulate on the surface of roots and leaves, but in soil contaminated areas very little PAH will be translocated from the roots to the shoots. Thus, for crops grown in contaminated soil, PAH concentrations are likely to be higher in or on the roots than in above-ground plant parts. The reverse can be true, however, in areas with high levels of fallout of PAH-contaminated particulate matter.

Plants appear to demonstrate the ability to metabolize PAH. This metabolism or degradation of PAH may take place regardless of the route of entry into the plant (leaves or roots). Plants including alfalfa, rye, orchard grass, and vetch were found to metabolize BaP that was assimilated by these plants (Durmishidze et al. 1974 as cited by Sims and Overcash 1983).

In summary, it has been demonstrated that plants may incorporate some PAH into their tissues. However, biomagnification levels are quite low. Coupled with low biomagnification levels and demonstrated metabolism of certain constituents of PAH, the potential for hazard to food chain consumers seems minimal.

Effects on Grazing Animals

While there is an extensive body of literature on the effects of PAH on laboratory animals, there is little data on the impacts to ruminant animals. Animals are often exposed to creosote from treated lumber and posts. Toxicosis of PAH from creosote is usually a result of exposure from chewing treated lumber (Reynolds and Stedelin 1982). In many cases the reported poisonings resulted from exposures in which pentachlorophenol was used in conjunction with creosote.

In a reported case of acute toxicosis (Reynolds and Stedelin 1982), a herd of Hereford cattle was grazed adjacent to a railroad tie treatment (creosote) plant. Heavy rains washed creosote into the pasture area and the cattle that were exposed to this wash-out event experienced weight loss, unthriftiness, and one death. Subsequent analyses identified matching creosote peaks in the HPLC (High Performance Liquid Chromatography) analyses for rumen content extracts for a water sample from the pasture and for a coal-tar creosote sample from a local lumber yard. While this incident does indicate an occurrence of acute toxicosis, it was most likely the result of water contaminated with creosote and forage which had creosote adsorbed into the foliage. Direct uptake of creosote from the soil by the plants and subsequent uptake by the cattle was probably not the pathway of poisoning in this incident.

Bioaccumulation of PAH from water in vertebrate organisms is considered to be short-term, unlike the long-term bioaccumulation that has been demonstrated for the persistent chlorinated organics (Versar, Inc. 1979). Numerous studies show that despite a high lipid solubility, PAH show little tendency for bioaccumulation in the fatty tissues of animals or man (EPA 1980a). Metabolism and excretion of PAH in the urine and feces of laboratory animals has been documented (Versar, Inc. 1979) further indicating that bioaccumulation is limited. The above discussion seems to indicate, therefore, that although uptake of PAH by cattle from existing contaminated forage and soil is possible, long-term accumulation of PAH appears unlikely.

It is known that PAH are metabolized, mainly in the liver, by mammals and humans. The metabolism takes place biochemically by the cytochrome P-450-dependent microsomal mixed-function oxidase (MFO) system. The MFO system can metabolize certain PAH and forms reactive epoxide metabolites which have been implicated as carcinogenic in experimental mammals (Sims and Grover 1974; Sims 1976; Lehr et al. 1978; and others).

In addition to the above pathway, a second pathway for formation of carcinogenic metabolites is the conversion of PAH epoxide metabolites by epoxide hydrase to vicinal glycols which may lead to carcinogenic bioactivation.

Although no acute or chronic toxicity to cattle from eating grass and soil contaminated with PAH is expected, the low levels ingested were evaluated as a carcinogenic risk. Cases of cancer in cattle from PAH uptake are unknown and no ingestion limits have been set for cattle. However, considering the very low ambient water quality criteria set for humans, a very low dose may be sufficient to cause cancer in cattle if they were to ingest the contaminated grasses and soil over a long period of time. However, because of the limited life expectancy of cattle, it appears highly unlikely that most cattle could be exposed for a period of time long enough to experience a carcinogenic effect.

PAH Uptake by Waterfowl

Many studies have been conducted on both the acute (short-term) and chronic (long-term) effects of crude and refined oils on waterfowl. Many studies also have been conducted in regards to ingestion of oil and oil products by waterfowl. In studies with mallard ducks (Anas platyrhynchos), Lawler et al. (1978) demonstrated accumulation of two and three-ring aromatic hydrocarbons into tissues from crude oil administered orally. The skin and underlying fat tissue accumulated aromatic hydrocarbons greater than the liver, breast muscle, heart muscle, brain, and blood. Furthermore, aromatic hydrocarbons did not accumulate in the same relative concentrations as found in crude oil suggesting differential uptake in metabolism.

Szaro et al. (1978) found that mallard ducklings fed 0.025, 0.25, 2.5, and 5.0 percent crude oil in the feed had evidence of liver and spleen pathology. In addition, behavior of ducklings was affected, as measured by avoidance responses, at the 2.5 and 5 percent oil diets. At even lower levels of oil diet (0.025 and 0.25 percent), plasma enzymes were elevated which indicated kidney and liver functions that are indicative of impending histopathology.

Hoffman and Gay (1981) studied the embryotoxic effects of BaP, chrysene and 7,12-dimethylbenz(a)anthracene in mallard ducks. These studies were conducted by externally applying PAH to the shell of an egg, and showed that PAH at relatively small levels are toxic to mallard embryos.

Belisle et al. (1981) found that both aliphatic and aromatic compounds (including naphthalene, methylnaphthalenes, fluorene, and phenanthrene) were found to accumulate in mallard eggs when the female diet contained 25,000 parts per million (ppm) petroleum. However, the residue levels of the aliphatic compounds were greater than those of aromatics.

Patton and Dieter (1980) studied the effects of petroleum hydrocarbons on hepatic (liver) functions of adult mallard ducks. One group of ducks was fed an ad libitum diet of breeder

mash containing a 1 percent mixture of 0.96 percent (9600 ppm) paraffin and a 0.04 percent (400 ppm) aromatic hydrocarbon mixture. A second group was fed a 1 percent mixture of 0.60 percent (6,000 ppm paraffin) and 0.40 percent (4,000 ppm) aromatic hydrocarbons in the breeder mash. The aromatic hydrocarbons in the mash included acenaphthene, acenaphthylene, and phenanthrene. Body weight decreased in the groups fed aromatic hydrocarbons during the first two months but returned to pretreatment levels after five months of the study. This was thought to be a result of decreased food intake due to the noxious odor of the food. No mortality, no gross organ abnormality, or decrement in liver functional capacity was shown. These parameters indicated that petroleum hydrocarbons produced little change in hepatic cells.

This data (Patton and Dieter 1980) suggested that the adult mallard is able to tolerate high concentrations of petroleum hydrocarbons for an extended period of time. The liver function test results suggested a dose-response relationship with the level of aromatic hydrocarbons in the feed, and though an increased physiological demand on the adult ducks was evident, the duck was able to tolerate high concentrations of these aromatic hydrocarbons for extended periods of time.

The only known study of direct uptake of PAH by waterfowl from food is that of Tarshis and Rattner (1982). In this study, crayfish, which were artificially contaminated with 14C-naphthalene, were force-fed to one year redhead ducks to determine accumulation of the 14C-naphthalene. The crayfish contained approximately 45 ppm (mg/kg) naphthalene wet weight providing a daily dose of 1.25 mg/bird. Greatest concentrations of 14C-naphthalene were found in the fat, gallbladder, and bile and kidney tissues. While there was appreciable accumulation of 14C-naphthalene, the exposure did not cause any overt signs of toxicity in the duck.

E. Reported Levels

in soils (mg/kg):		
in oak forest, MA	0.04	Blumer 1961
in pine forest, MA	0.04	Blumer 1961
garden soil, MA	0.09	Blumer 1961
mixed forest, CT	0.24	Blumer 1961
plowed field, CT	0.90	Blumer 1961
mixed forest, MA	1.30	Blumer 1961
near highway	2.0	White and Vanderslice 1980
urban soil	50-75	White and Vanderslice 1980

in sediments (mg/kg):		
Burdekin R./G B reef	.0008	Smith et al 1985
Pskov region	.0010	Ershova, 1968
Heron I./Queensland	.0025	Smith et al 1985
River Sunzha	.0092	Samoilovich et al. 1968
River Sunzha	.0190	Samoilovich et al. 1968

Ross River/G B reef	.0390	Smith et al 1985
Rublevskoye Res.	.0440	Il'nitskii et al.1972
Buzzards Bay, New Bedford, MA	.0750	Giger and Blumer, 1974
Penobscot Bay, ME, outer region	.0971	Johnson et al. 1985
River Seine	.1000	Mallet, 1965
New York Bight	.1426	Reid et al. 1982
USA/general	.2225	in Smith et al 1985
Japan/general	.2250	in Smith et al 1985
Grosser Ploner See	.2600	Grimmer et al. 1975
Penobscot Bay, ME, inner region	.3304	Johnson et al. 1985
Buzzards Bay, New Bedford, MA	.3700	Giger and Blumer, 1974
Khiminskoe Re.	.3900	Il'nitskii et al.1972
River Seine	.3900	Mallet, 1965
France/general	.4000	in Smith et al 1985
Boston Harbor	.4300	Mass DEQE 1985
Lake Constance	.4430	Muller et al. 1977
Commencement Bay, WA	.5000	Malins et al. 1982
Gladstone H./Queensland	.8200	Smith et al 1985
Townsville H./Queensland	.8528	Smith et al 1985
Newton Creek NY	1	Anderson 1982
Boston Harbor	1.1739	Shiaris and Jambard-Sweet 1986
Long Island Sound	1.3000	Reid et al. 1982
The Graves, Boston MA	1.5770	Shiaris and Jambard-Sweet 1986
Grosser Ploner See	1.6100	Grimmer et al. 1975
Elliot Bay Pier54, WA	2	Malins et al. 1982
Australia/general	2.6000	in Smith et al 1985
Italy/general	3	in Smith et al 1985
River Seine	3	Mallet, 1965
Mystic River Boston MA	6.5000	Mass DEQE 1985
USSR River	8.2000	Fedorenko, 1964
River Seine	8.5000	Mallet, 1965
Elizabeth River VA	9	Bieri,et al.(in press)
River Seine	10	Mallet, 1965
River Usk	11	Eglinton et al. 1975
Chelsea River, MA	13.4620	Shiaris and Jambard-Sweet 1986
River Seine	15	Depuis, 1960
USSR River	17	Fedorenko, 1964
Boston Harbor Aquarium/Fort Point	48.4665	Shiaris and Jambard-Sweet 1986

in air (ng/m3):

in cities	0.03-4	(winter)	USEPA (1979)
in cities	1.3-18.5	(annual)	USEPA (1979)
Lake Michigan	0.3-1.8		Radian Corp.(1983)
Urban NJ summer	0.19-0.42		Radian Corp.(1983)
resid. (Florence AL)	<0.34-7.24		Radian Corp.(1983)
rural (Florence AL)	<0.27-1.82		Radian Corp.(1983)
urban winter	0.6-104		IARC(1973)
urban summer	0.03-4		IARC(1973)
Chatanooga TN	1.3		Radian Corp.(1983)

Jefferson County AL	3.31	Radian Corp.(1983)
Birmingham AL	0.57	Radian Corp.(1983)
U.S. cities	0.13-3.2	Colucci and Begeman 1971
U.S. cities	1.3-18.5	Sawicki et al. 1962

New York State monitors BaP at various upstate sites. The maximum ambient concentrations were 2.2, 2.1, and 4.5 ng/m3 in 1982, 1983, and 1984; values were typically less than 1 ng/m3. Hunt et al (1984) report that regional BaP levels for NYS are on the order of 0.4 ng/m3 for rural areas.

in drinking water (ng/l):

Fairborn, OH	0.1	Basu and Saxena 1977
Edicott + Buffalo NY	0.2	Basu and Saxena 1977
Philadelphia PA	0.3	Basu and Saxena 1977
Appleton, WI	0.4	Basu and Saxena 1977
New York, NY	0.5	Basu and Saxena 1977
New Orleans, LA	1.6	Basu and Saxena 1977
Wheeling, WV	2.1	Basu and Saxena 1977

in surface waters (ng/l):

River Danube at Ulm	0.6	Borneff and Kunte 1964
Estimated Interquartile range for Rivers and Lakes	1 - 100	based on White and Vanderslice 1980
River Main	2.4	Borneff and Kunte 1964
Delaware R., PA	4.1	Basu and Saxena 1978
Ohio R., WV	5.6	Basu and Saxana 1978
River Gersprenz	9.6	Borneff and Kunte 1964
River Schussen	10	Borneff and Kunte 1964
River Aach	17	Borneff and Kunte 1964
Monongahela R., PA	42 - 77	Basu and Saxana 1978
Rhine River	80	Borneff and Kunte 1964

F. National Guidelines and Standards

Water Quality: U.S. EPA (1980) has recommended criteria related to specific incremental lifetime risk levels of 10E-5, 10E-6, and 10E-7. The corresponding water concentrations assuming ingestion of water and aquatic organisms are 28 ng/l, 2.8 ng/l, and 0.28 ng/l respectively. If the above estimates are made for consumption of aquatic organisms only, the levels are 311 ng/l, 31.1 ng/l, and 3.11 ng/l respectively. These values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to B[a]P. It should be noted that the carcinogenic potency of B[a]P and other PAH compounds is currently being reevaluated by a number of groups. With regard to the protection of aquatic life, no numerical criteria have been developed.

OSHA Limit: an 8-hour TWA concentration limit of 0.2 mg/m3 has been set for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, B[a]P, phenanthrene, acridine, chrysene, and pyrene).

NIOSH: recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m3 of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA.

G. Selected State Guidelines

N.Y. State has a "non-detectable" groundwater quality standard for B[a]P by a specified testing method. A guidance value of 2 ng/l is specified by the State for surface waters with regard to the protection of human health. N.Y. State also had an Acceptable Ambient Level (AAL) of 33 ng/m3 for B[a]P in air. However, this AAL has not been included in recent documents published by the State.

Minnesota Department of Health has developed a limitation of 28 ng/l for the sum of carcinogenic PAH and this would include B[a]P.

A number of states have general water quality criteria that address toxic substances but numerical criteria are often not established.

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BENZ(a)ANTHRACENE
[Benzo (a)anthracene; 1,2-Benzo(a)anthracente;
Benzo (a)phenanthrene]

A. Physical and Chemical Properties

Property	Value	Reference
Molecular wt.	228.3 g/mole	
Water Solubility	5.7×10^{-3} ppm @ 20°C	Mabey et al. 1982
Vapor Pressure	2.20×10^{-8} torr @ 20°C	Mabey et al. 1982
Koc	2.00×10^{5}	Mabey et al. 1982
Kow	4.10×10^{5}	Mabey et al. 1982
Hc	1.00×10^{-6} atm-m^{3}/mole	Mabey et al. 1982

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

Several studies indicate that benz(a)anthracene is carcinogenic in animals, and IARC has evaluated that evidence as sufficient to establish the carcinogenicity of benz(a)anthracene in animals (IARC 1983). A CPF has not been developed by the U.S. EPA Carcinogen Assessment Group. Based on the work of Bingham and Falk (1969), Clement Associates (1987) estimated that benz(a)anthracene has a relative potency to benzo(a)pyrene of 0.145. (Potency is approximately 14.5% of that of benzo(a)pyrene.) This value can be used in the relative potency approach for estimating carcinogenic risk.

Tumors developed at incidences of 8/31, 4/20, 11/21, and 17/22 when dodecane was used at a vehicle (Bingham and Falk 1969). In a comparison of the carcinogenic effects of benzo(a)pyrene and benz(a)anthracene, a 0.05% solution of benzo(a)pyrene in dodecane applied weekly produced tumors in 10/10 animals in 11 weeks while a 0.2% solution of benz(a)anthracene produced tumors in 11/21 animals in an average of 61 weeks. A 1% solution of

benz(a)anthracene produced tumors in 17/22 animals in an average of 42 weeks. A skin tumor (papilloma) incidence of 100% was reported in 17 mice painted with 0.05% benz(a)anthracene in acetone (Graffi et al 1953 as cited in IARC 1973).

Extensive testing for mutagenicity has been documented (IARC, 1983).

C. Pharmacokinetics

Some benz(a)anthracene metabolites have been shown to induce mutations, cell transformation, and to bind to nucleic acids. The metabolites 3,4-diol-1,2-epoxide and 3,4-dihydrodiol, are mutagenic and tumorigenic (Sims and Grover 1974, 1981; Conney 1982 as cited in IARC 1983).

Nucleic acid (DNA) adducts are formed in the skin from the metabolites 3,4-diol-1,2-epoxide and 8,9-diol-10,11-epoxide (Sims and Grover 1974, 1981; Conney 1982 as cited in IARC 1983).

Benz(a)anthracene induced benzo(a)pyrene hydroxylase in rat placenta (Welch et al. 1969 as cited in IARC 1983).

No information is available regarding dermal or oral absorption coefficients, although benz(a)anthracene was reported to be readily transported across the gastrointestinal mucosa (Rees et al. 1971 as cited in U.S. EPA 1984).

D. Environmental Effects

Benzo(a)anthracene is expected to be similar to other higher molecular weight PAH compounds with regard to effects on aquatic and terrestrial biota. A general review of such effects is provided in the profile for benzo(a)pyrene.

Hinga et al (1980) examined the biogeochemistry of C-14 labelled benz(a)anthracene in an enclosed marine ecosystem. The experiment was conducted for 230 days. At the end of the experiment, 29% of the chemical had been respired to CO_2, while the remaining extractable activity (43%) was evenly divided between parent compound and intermediate metabolic products. Total C-14 activity was removed from the water with a half-life of about 52 hours, while the C-14 parent comound had a half-life of 24 hours. The chemical became associated with the sediments and was mixed deeper into the sediments by benthic animal activity. The authors made a rough calculation of the half-life in sediments and noted that half-lives on the order of 1.2 -3 years may be calculated. They further point out, however, that the occurrence of benz(a)anthracene at some depth in natural sediments suggests that a fraction of the compound and perhaps some of its metabolites may persist indefinitely.

E. Guidelines & Standards

Water quality: U.S. EPA (1986) has recommended criteria related to specific incremental lifetime risk levels of 10-5, 10-6, and 10-7. The corresponding water concentrations assuming ingestion of water and aquatic organisms are 28, 2.8, and 0.28 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, the levels are 311, 31.1, and 3.11 ng/l respectively. These values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to B(a)P. The carcinogenic potency of B(a)P and other PAH compounds is currently being re-evaluated by several industry groups and EPA. With regard to the protection of aquatic life, no numerical criteria have been developed.

OSHA Limit: An 8-hour TWA concentration limit of 0.2 mg/m^3 has been set for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene, and pyrene).

NIOSH: Recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA.

F. Reported Values

In Soil:	Concentration mg/kg	Reference (see Appendix A)
in forest	0.005-0.02	White and Vanderslice 1980 and IARC, 1973
near traffic	1.5	
urban soil	20.0	" "

In Sediment

Burdekin R./G B reef	.0023	Smith et al. 1985
Heron Island/Queensland	.0045	Smith et al. 1985
Ross R./G B reef	.0190	Smith et al. 1985
Buzzards Bay, New Bedford, MA	.0410	Giger and Blumer 1974
Penobscot Bay, ME, outer region	.0970	Johnson et al. 1985
Japan/general	.1390	Smith et al. 1985
New York Bight	.1704	Reid et al. 1982
Gladstone H./Queensland	.1800	Smith et al. 1985
USA/general	.1855	Smith et al. 1985
Penobscot Bay, ME, inner region	.3140	Johnson et al. 1985
Buzzards Bay, New Bedford, MA	.3300	Giger and Blumer, 1974
Townsville H./Queensland	.4834	Smith et al. 1985
Boston Harbor	1.1425	MA DEQE 1985
Australia/general	1.7000	Smith et al. 1985
Long Island Sound	2.2100	Reid et al. 1982
Commencement Bay, WA	4	Malins et al. 1982
Newton Creek, NY	6	Anderson 1982
Elliot Bay Pier54, WA	7	Malins et al 1982
Elizabeth River VA	11	Bieri et al. (in press)
Mystic River, Boston MA	11.4	MA DEQE 1985

In Air:	(ng/m^3)	
Residential (Florence, AL)	0.04-5.5	Radian Corporation 1983
Rural (Florence, AL)	0.07-0.6	Radian Corporation 1983
Urban NJ (summer)	0.07-0.16	White & Vanderslice 1980
Urban	0.6-316	
U.S. Cities	0.18-4.6	Fox & Stanley 1980 Gordon 1976
Siena, Italy (summer)	1.6	White & Vanderslice 1980
Siena, Italy (winter)	94	White & Vanderslice 1980
Pittsburg (summer)	136	White & Vanderslice 1980

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U.S. Environmental Protection Agency. 19865 (and updates). Ambient Water Quality Ctriteria for Polycyclic Aromatic Hydrocarbons in the Environment Washington, D.C.

BENZO(k)FLUORANTHENE
[11,12-Benzo(k)fluoranthene]

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	252.3 g/mol	
Water Solubility	4.3×10^{-3} ppm at 25°C	Mabey et al. 1982
Vapor Pressure	5.0×10^{-7} torr at 20°C	Mabey et al. 1982
Koc	5.5×10^{5}	Mabey et al. 1982
Kow	1.15×10^{6}	Mabey et al. 1982
Kh	negligible	Mabey et al. 1982
Hc	3.87×10^{-5} atm-m^{3}/mol	Mabey et al. 1982

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

IARC concluded that there is sufficient evidence for the carcinogenicity of benzo(k)fluoranthene in experimental animals. Benzo(k)fluoranthene has been administered by skin painting, subcutaneous injection, and intrapulmonary injection.

Mutations were induced in Salmonella typhimurium strains TA100 and TA98 in the presence of a metabolic activation system from Aroclor-induced rat liver (LaVoie et al., 1980; Herman et al., 1980, as cited in IARC 1983).

There are no data available regarding other toxicological end-points including reproductive effects and teratogenicity.

Although IARC has identified sufficient evidence for its carcinogenicity, there is no Unit Cancer Risk (UCR) available for benzo(k)fluoranthene. Thus, a target concentration cannot be calculated at this time. Based on the work of Deutsch-Wenzel et al., (1983), Clement Associates (1987) estimated that the potency of benzo(k)fluoranthene - relative to benzo(a)pyrene - is approximately 0.066. This number can be used in the relative potency approach.

C. Pharmacokinetics

A mutagenic metabolite of benzo(k)fluoranthene is the 8,9-dihydrodiol (LaVoie et al., 1980, as cited in IRAC, 1983). No information was available regarding DNA adduct formation or absorption factors.

D. Environmental Effects

No specific information was obtained on the environmental effects of this PAH to freshwater animals. As it is an animal positive coarcinogen, it can be expected to have potential carcinogenic/mutagenic effects on organisms in the environment.

E. Reported Levels

In Sediments:	Concentration mg/kg	Reference
Burdekin R./G B reef	.0001	Smith et al 1985
Heron I./Queensland	.0003	Smith et al 1985
Ross R./G B reef	.0025	Smith et al 1985
Gladstone H./Queensland	.0160	Smith et al 1985
Townsville H./Queensland	.0654	Smith et al 1985
Penobscot Bay, ME, outer region	.1008	Johnson et al., 1985
Japan/general	.1800	Smith et al., 1985
Australia/general	.2000	Smith et al., 1985
Boston Harbor	.3083	MA DEQE, 1985
Penobscot Bay, ME, inner region	.3100	Johnson et al. 1985
Mystic River, Boston MA	8.5000	MA DEQE, 1985
In Drinking Water:	**ng/l**	
Ranges for ten cities	ND-0.7	Basu and Saxena (1978)

In Air:	(ng/m3)	
urban	0.03-1.3	White & Vanderslice 1980
urban	0.1-10	U.S. EPA 1982
U.S. cities	1.0-12.5	Sawicki et al., 1962
Hamilton, Ontari	0.87-1.4	Radian Corp. 1983
New York City	0.60	Radian Corp., 1983
urban, NJ (summer)	0.11-0.33	Radian Corp., 1983
residential (Florence, AL)	3.7-52.7	Radian Corp., 1983
rural (Florence, AL)	1.5-9.5	Radian Corp., 1983

F. National Guidelines

Water quality: U.S. EPA (1986) has recommended criteria related to specific incremental lifetime risk levels of 10-5, 10-6, and 10-7. The coressponding water concentrations assuming ingestion of water and aquatic organisms are 28, 2.8, and 0.28ng/l respectively. If the above estimates are made for consumption of aquatic organisms only, the levels are 311, 31.1, and 3.1 ng/l respectively. The values acknowledge the conservative assumption that all carcinogenic PAH's are equal in potency to Benzo(a)pyrene. The carcinogenic potency of B(a)P and other PAH's is currently being reevaluated by a number of groups. With regard to protectiion of aquatic life, no numerical criteria have been developed.

G. References/Bibliography

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U.S. Environmental Portection Agency. 1986 (and updates). Ambient Water Quality Criteria for Polycyclic Aromatic Hydrocarbons. EPA-440/5-80-69.

Wynder, E. L. and D. Hoffman. 1959. The carcinogenicity of benzofluoranthenes. Cancer. 12:1194-1199.

BENZO(g,h,i)PERYLENE
(1,12-Benzoperylene)

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	276.3 g/mol	Merck, 1983
Water solubility	2.6×10^{-4} ppm at 25°C	Verschueren, 1983
Vapor pressure	1.03×10^{-10} torr at 25°C	Mabey et al., 1982
Koc	1.6×10^{6}	" " "
Kow	3.2×10^{6}	" " "
Hc	1.21×10^{-7} atm-m^{3}/mol	U.S. EPA, 1982

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

IARC (1983) concluded that the available data are inadequate to evaluate the carcinogenic potential of benzo(g,h,i)perylene. Based on a study by Deutsch-Wenzel and others (1983), Clement Associates (1987) reported that the potency of this compound, relative to benzo(a)pyrene, was 0.022. This number can be used in the relative potency method.

Mutations due to benzo(g,h,i)perylene were evident in in vitro bacteria mutagenicity tests. The Ames test resulted in positive mutagenicity in Salmonella typhimurium strains TA100, TA98, TM677, and TA15318 (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979; and Kaden et al., 1979, as cited in IARC, 1983; and Gibson et al., 1978 as cited in U.S. EPA, 1982). The results were negative in Salmonella typhimurium strain TA1535

(Gibson et al., 1978 as cited in U.S. EPA, 1982). Cell transformation tests using hamster embryo cells in vitro yielded negative results (Quarles et al., 1979, as cited in IARC, 1983).

There are no data available on other toxic end-points including reproductive effects and teratogenicity.

Currently, there is no CPF available for this compound. Sufficient data to calculate an AAI have not been identified.

C. Pharmacokinetics

No data are available on the formation of carcinogenic metabolities, DNA adduct formation, enzyme induction or absorption.

D. Environmental Effects

The benzo(a)pyrene environmental profile discusses the general effects of PAHs.

E. Reported Levels

In sediment:

Location	Concentration (mg/kg)	Reference (see Appendix A)
Burdekin R./G B reef	.0003	Smith et al., 1985
Heron I./Queensland	.0039	" " "
Ross R./G B reef	.0063	" " "
Buzzards Bay, New Bedford, MA	.0660	Giger and Blumer, 1974
Penobscot Bay, ME, outer region	.1566	Johnson, et al., 1985
USA/general	.1780	Smith et al., 1985
Japan/general	.2000	" " "
Gladstone H./Queensland	.2000	" " "
Buzzards Bay, New Bedford, MA	.2800	Giger and Blumer, 1974
Penobscot Bay, ME, inner region	.4291	Johnson et al., 1985
Townsville H./Queensland	.4493	Smith et al., 1985
Savern Estuary, UK	.9	John et al., 1979
Australia/general	1.5000	Smith et al., 1985

In soils:		
in urban soils	100	Radian Corp., 1983
In drinking water:	(ng/l)	
Ten city systems	0.4 - 4.0	Basu and Saxena, 1978
In Air:	(ng/m^3)	
U.S. cities	0.2-9.2	Gordon and Bryan, 1973
U.S. cities	5.1-21.3	Sawicki et al., 1962
	0.2-50	U.S. EPA, 1982
New York City	0.90	Radian Corp., 1983
Hamilton Ontario	5.2-13	" " "
urban NJ (summer)	0.57-0.75	" " "

F. Guidelines and Standards

Water quality: U.S. EPA (1982) has recommended criteria related to specific incremental lifetime risk levels of 10-5, 10-6, and 10-7. The corresponding water concentrations, assuming ingestion of water and aquatic organisms, are 28, 2.8 and 0.28 ng/l, respectively. If these estimates are made for the consumption of aquatic organisms only, the levels are 311, 31.1 and 3.1 ng/l, respectively. The values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to benzo(a)pyrene. The carcinogenic potency of B(a)P and other PAHs is currently being re-evaluated by several industry groups and EPA. With regard to the protection of aquatic life, no numerical criteria have been developed.

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U.S. Environmental Protection Agency. 1982. An Exposure and Risk Assessment for Benzo(a)pyrene and Other Polycyclic Aromatic Hydrocarbons. Vol. IV. Final Draft Report. Office of Requlations and Standards.

Van Duuren, B.L., C. Katz, and B.M. Goldschmidt. 1973. Brief communication: cocarcinogenic agents in tobacco carcinogenesis. J. Natl. Cancer Inst. 51:703-705.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. Second Edition. Van Nostrand Reinhold, NY.

CHRYSENE

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	228.3 g/mol	Verschueren, 1983
Water solubility	6.0×10^{-3} ppm at 25°	" "
Vapor pressure	6.9×10^{-9} torr at 25°C	Mabey et al., 1982
Koc	2.0×10^{5}	" " "
Kow	4.1×10^{5}	" " "
Hc	1.05×10^{-6} atm-m^{3}/mol	" " "

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

There are no data on the toxicity of chrysene to man (IARC, 1983). The relative tumorigenic potency of chrysene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays tested using similar protocols (U.S. EPA, 1984). The ranking was as follows: benzo(a)pyrene > dibenzo(a,h)anthracene > benzo(b)fluoranthene > benzo(a)anthracene > indeno(1,2,3-c,d)pyrene > chrysene.

There is limited evidence that chrysene is mutagenic in short-term assays (IARC, 1983). There is no experimental data on the teratogenicity of chrysene in mammals.

IARC (1983) concluded that there is limited evidence to indicate that chrysene is carcinogenic in experimental animals. EPA's CAG has not estimated a CPF for chrysene (U.S. EPA, 1985). Based on the data of Wynder and Hoffman (1959), Clement Associates (1987)

estimated that chrysene had a relative potency, to B(a)P, of approximately 0.0044. This number can be used in the relative potency method.

C. Pharmacokinetics

There are no pharmacokinetic data for chrysene in man (U.S. EPA, 1980).In general, many PAHs can produce toxicity after inhalation, oral or dermal exposure. Thus, it is believed that PAHs are absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed-function-oxidase system, and elimination most likely occurs through the hepatobiliary tract.

Several monohydroxyl and dihydrodiol derivatives of chrysene have been reported (Sims, 1970; Nordquist et al., 1981; and Jacob et al., 1982, as cited in IARC, 1983). Epoxides of the 1,2-dihydrodiol and 3,4-dihydrodiol have also been reported (Chou et al., 1981; Nordquist et al., 1981; and Vyas et al., 1982, as cited in IARC, 1983). The 1,2-dihydrodiol and 1,2-diol-3,4-epoxide have been shown to be mutagenic in bacterial and mammalian cells (Wood et al., 1977; 1979, as cited in IARC, 1983) and inducers of pulmonary adenomas in newborn mice (Buening et al., 1979; and Chang et al., 1983, as cited in IARC, 1983). In addition, the 1,2-dihydrodiol has been shown to be a tumor-initiating agent on mouse skin (Levin et al., 1978; Slags et al., 1980; and Chang et al., 1983, as cited in IARC, 1983). The 1,2-diol-3,4-epoxide is believed to be the metabolite of chrysene that forms adducts with DNA (Hodgson et al., 1982; and Vigny et al., 1982, as cited in IARC, 1983).

D. Environmental Effects

The B(a)P environmental profile discusses the general environmental effects of PAHs.

As with many other individual PAHs, no specific information was obtained on the aquatic toxicity of this chemical. However, it is an animal-positive carcinogen. Chrysene produced skin tumors in mice following repeated paintings. High doses (2-20 mg) given by subcutaneous injection to mice produced a low incidence of tumors. Thus, chrysene, like other PAHs, may have carcinogenic or mutagenic effects on aquatic and terrestrial organisms. Adequate data for the charcterization of toxicity in domestic animals or wildlife is not available.

E. Reported Levels

In soil:	Concentration (mg/kg)	Reference (see Appendix A)
(unspecified)	0.04-0.24	IARC, 1973
urban	20	White and Vanderslice, 1980

In sediments:	(mg/kg)	
Heron I./Queensland	.0017	Smith et al., 1985
Burdekin R./G B reef	.0036	" " "
Gladstone H./Queensland	.0110	" " "
Ross R./G B reef	.0150	" " "
Buzzards Bay, New Bedford, MA	.0400	Giger and Blumer, 1974
Penobscot Bay, ME, outer region	.0598	Johnson et al., 1985
USA/general	.1400	Smith et al., 1985
New York Bight	.1409	Reid et al., 1982
Penobscot Bay, ME, inner region	.1969	Johnson et al., 1985
Buzzards Bay, New Bedford, MA	.2400	Giger and Blumer, 1974
Townsville H./Queensland	.4577	Smith et al., 1985
Boston Harbor	.7840	MA DEQE, 1985
Australia/general	1.5000	Smith et al., 1985
Long Island Sound	1.6685	Reid et al., 1982
Savern Estuary	2.9	John et al., 1979
Newton Creek, NY	3	Anderson, 1982
Commencement Bay, WA	4	Malins et al., 1982
Elliot Bay Pier 54, WA	6	" " "
Mystic River Boston, MA	9.5000	MA DEQE, 1985
Elizabeth River, VA	19	Bieri et al. (In press)

In air:	(ng/m^3)	
(unspecified)	0.2-10	U.S. EPA, 1982
urban areas	0.6-4.8	White and Vanderslice, 1980
U.S. cities	0.2-6.4	Gordon and Bryan, 1973
urban (summer)	2.5-3.6	IARC, 1973
urban (winter)	2.0-361	" "

F. Guidelines and Standards

Water quality: U.S. EPA (1986) has recommended criteria related to specific incremental lifetime risk levels of 10-5, 10-6 and 10-7. The corresponding water concentrations assuming ingestion of water and aquatic organisms, are 28, 2.8 and 0.28 ng/l, respectively. If these estimates are made for the consumption of aquatic organisms only, the levels are 311, 31.1 and 3.1 ng/l, respectively. The values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to B(a)P. The carcinogenic potency of B(a)P and other PAHs is currently being re-evaluated by several industry groups and EPA. With regard to the protection of aquatic life, no numerical criteria have been developed.

OSHA limit: An 8-hour TWA concentration limit of 0.2 mg/m^3 has been set for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene and pyrene).

NIOSH recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample determined as a 10-hour TWA.

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Wynder, E.L., and D. Hoffman. 1959. A study of tobacco carcinogenesis. VII. The role of higher polycyclic hydrocarbons. Cancer 12:1079-1086.

DIBENZO(a,h)ANTHRACENE
[1,2:5,6-Dibenzanthracene, Dibenz(a,h)anthracene]

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	278.36 g/mol	Mabey et al., 1982
Water solubility	5.0×10^{-4} ppm at 25°	" " "
Vapor pressure	1.0×10^{-10} torr at $20^{\circ}C$	" " "
Koc	3.3×10^{6}	" " "
Kow	6.9×10^{6}	" " "
Hc	7.30×10^{-8} atm-m^3/mol	" " "

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

Dibenzo(a,h)anthracene [DB(a,h)A] has been tested for carcinogenicity in a variety of test species using different routes of exposure; positive results have been reported in the majority of studies. Little data were identified concerning toxic effects other than tumor induction in the test species. IARC (1983) has determined that there is sufficient evidence to conclude that DB(a,h)A is carcinogenic to experimental animals.

DB(a,h)A has been tested in various short-term test systems with the majority reporting positive results (IARC, 1983). The compound showed positive evidence for mutagenicity when tested in *Salmonella typhimurian* strains TA100 and TA98 after activation with rat liver microsomal fractions. Positive results were also reported in a test for DNA damage in *Escheria coli*, these tests also employed activation with exogenous metabolic systems (IARC, 1983).

The induction of unscheduled DNA synthesis was also reported in human epithelial and HeLa cells exposed in vitro, the latter using a matabolic activation system. The 1,2-, 3,4- and 5,6-dihydrodiols, considered probable metabolites of DB(a,h)A, were tested for mutagenicity in bacteria in the presence of rat liver preparations and the 3,4-dihydrodiol was found to be the most mutagenic. When tested in hamster embryo cells, the 5,6-oxide was reported to cause significant cell transformation (IARC, 1983).

Based on the work of Wynder and Hoffman (1959), Clement Associates (1987) estimated that DB(a,h)A had a relative potency, to benzo(a)pyrene, of 1.11. This number can be used in the relative potency method.

C. Pharmacokinetics

No quantitative data were located concerning the absorption of DB(a,h)A in experimental animals. The 5,6-oxide and the 1,2-, 3,4- and 5,6-dihydrodiols have been detected as metabolites of DB(a,h)A after incubation in rat liver preparations (Selkirket et al., 1971; and MacNicoll et al., 1979, 1980, as cited in IARC, 1983) and mouse skin in organ culture (MacNicoll et al., 1980, as cited in IARC, 1983). The 5,6-oxide was found to bind to cellular macromolecules in mammalian cells (Kuroki, et al., 1972 as cited in IARC, 1983). Nucleoside adducts have been detected in mouse skin following the topical application of DB(a,h)A, but were not characterized (Phillips et al., 1979, as cited in IARC, 1983).

No information on the tissue distribution or excretion of DB(a,h)A could be located.

D. Environmental Effects

The benzo(a)pyrene environmental profile discusses the general effects of PAHs.

E. Reported Levels

In soil:	Concentration (mg/kg)	Reference (see Appendix A)
Iceland	0.0023	IARC, 1973
air field	0.351	" "

In sediment:		
Penobscot Bay, ME, outer region	.0164	Johnson et al., 1985
Penobscot Bay, ME, inner region	.0435	" " "
Severn Estuary	.97	John et al., 1979
In air:	ng/m^3	
urban	3.2-32	IARC, 1973

F. Guidelines and Standards

Water quality: U.S. EPA (1986) has recommended criteria related to specific incremental lifetime risk levels of 10^{-5}, 10^{-6} and 10^{-7}. The corresponding water concentrations, assuming the ingestion of water and aquatic organisms, are 28, 2.8 and 0.28 ng/l, respectively. If these estimates are made for consumption of aquatic organisms only, the levels are 311, 31.1 and 3.1 ng/l, respectively. The values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to B(a)P. The carcinogenic potency of B(a)P and other PAHs is currently being re-evaluated by several industry groups and EPA. With regard to the protection of aquatic life, no numerical criteria have been developed.

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U.S. Environmental Protection Agency. 1982. An Exposure and Risk Assessment for Benzo(a)pyrene and Other Polycyclic Aromatic Hydrocarbons. Vol. IV. Final Draft Report. Office of Water Regulations and Standards (WH-553), Washington, D.C.

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Wynder, E.L., and D. Hoffman. 1959. A study of tobacco carcinogenesis. VII. The role of higher polycyclic hydrocarbons. Cancer 12:1079-1086.

INDENO(1,2,3-c,d)PYRENE
(2,3-Phenylenepyrene; 2,3-o-Phenylenepyrene)

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	276.34 g/mol	Verschueren, 1983
Water solubility	6.20×10^{-1} ppm at 25°C	U.S. EPA, 1980
Vapor pressure	1.0×10^{-10} torr at 20°C	" "
Koc	1.6×10^{6}	Mabey et. al., 1982
Kow	3.2×10^{6}	" " "
Hc	6.95×10^{-8} atm-m^3/mol at 20°C	" " "

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effect to Humans

There are no data on the toxicity of indeno(1,2,3-c,d)pyrene to man (IARC, 1983).

The relative tumorigenic potency of indeno(1,2,3-c,d)pyrene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays conducted using similar protocols (U.S. EPA, 1984). The ranking was as follows: B(a)P > dibenzo(a,h)anthracene > benzo(b)fluoranthene > benzo(a)anthracene > indeno(1,2,3-c,d)pyrene > chrysene.

Indeno(1,2,3-c,d)pyrene induced mutations in Salmonella typhimurium strain TA100 at a concentration of 20 ug/plate, and in strain TA98 at a concentration of 2 ug/plate in the presence of an exogenous metabolic activating system (IARC, 1983). Owing

to the lack of mutagenicity testing in other systems, IARC (1983) considered the available evidence inadequate to classify indeno(1,2,3-c,d)pyrene as a mutagen.

IARC (1983) did not identify studies which evaluated the effect of indeno(1,2,3-c,d)pyrene exposure on reproductive function, teratogenicity or systemic toxicity.

IARC (1983) concluded that there is sufficient evidence to suggest that indeno(1,2,3-c,d)pyrene is carcinogenic in experimental animals and recommended that it should be regarded as carcinogenic in man. At this time, EPA's CAG has not estimated a CPF for indeno(1,2,3-c,d)pyrene (U.S. EPA, 1985).

Based on a study by Deutsch-Wenzel and others (1983), Clement Associates (1987) estimated that indeno(1,2,3-c,d)pyrene has a relative potency, to B(a)P, of 0.232. This value can be used in the relative potency method.

C. Pharmacokinetics

There are no pharmacokinetic data of indeno(1,2,3-c,d)pyrene in man (U.S. EPA, 1980). In general, many PAHs can produce toxicity after inhalation, oral or dermal exposure. Thus, it is believed that they are readily absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed-function-oxidase system and eliminated primarily through the hepatobiliary tract.

D. Environmental Effects

The B(a)P environmental profile discusses the general effects of PAHs.

E. Reported Levels

In air:	Concentration (ng/m^3)	Reference (see Appendix A)
U.S. cities	0.03-1.34	Gordon, 1976; Gordon and Bryan, 1973
urban NJ (summer)	0.49-11	Radian Corp., 1983

In drinking water:	(ng/l)	
Range for ten cities	ND - 2.2	Basu and Saxena, 1978

F. Guidelines and Standards

Water quality: U.S. EPA (1980; 1986) has recommended criteria related to specific incremental lifetime risk levels of 10^{-5}, 10^{-6} and 10^{-7}. The corresponding water concentrations, assuming ingestion of water and aquatic organisms are 28, 2.8 and 0.28 ng/l, respectively. If these estimates are made for the consumption of aquatic organisms only, the levels are 311, 31.1 and 3.1 ng/l, respectively. The values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to B(a)P. The carcinogenic potency of B(a)P and other PAHs is currently being re-evaluated by several industry groups and EPA. With regard to the protection of aquatic life, no numerical criteria have been developed.

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NAPHTHALENE

Information presented below has been assembled to aid in the preparation of risk assessments. Detailed data on health end environmental effects can be found in the bibliography provided at the end of this profile.

A. Physical and Chemical Properties

Property (a)	Value	Reference
Molecular wt.	128.2	Mabey et al. 1982
Water solubility	31.7 ppm @ 25oC	Mabey et al. 1982
Vapor pressure	4.92 x 10-2 mm Hg @ 20oC	USEPA 1980a
	8.70 x 10-2 mm Hg @ 25oC	Mabey et al. 1982
Koc	9.40 x 10E2	Mabey et al. 1982
Kow	1.95 x 10E3	Mabey et al. 1982
Kh	negligible	Mabey et al. 1982
Hc	4.60 x 10-4 atm. m3/mole	Mabey et al. 1982

aKoc = Organic carbon-water partition coefficient; Kow = octanol-water partition coefficient; Kh = hydrolysis rate constant; Hc = Henry's law constant

B. Toxic Effects to Humans

At present, for the purposes of risk assessments, naphthalene is treated as a non-carcinogen. This means that a threshold "acceptable daily intake" value can be used as a basis for comparison with estimated exposure concentrations.

In humans, exposure to sufficient concentrations of naphthalene through inhalation, ingestion, or dermal contact may cause intravascular hemolysis or the less severe symptoms of eye irritation, headache, confusion, tremors, nausea, vomiting, abdominal pain, and bladder irritation (Sittig 1985). In severe cases hematological effects have included red cell fragmentation, icterus, severe anemia, leukocytosis and dramatic decreases in hemoglobin, hemacrit, and red cell counts. Hemolysis can also lead to renal disease from precipitated hemoglobin (USEPA 1982) Poisonings have occurred in humans as a result of the ingestion of moth balls as well as from clothing infants in materials that had been stored in moth balls. A study of workers exposed to naphthalene for a period of 5 years found corneal ulceration,

cataracts, and some lenticular and general opacities in 8 of the 21 employees examined (Ghetti and Mariani 1956 as cited in Sandmeyer 1981).

The dose of 53 mg/kg/day from the subchronic study described by Shopp et al. (1984) can be used as a NOAEL for calculating on ADI. A safety factor of 1000 is applied to this to account for the use of a subchronic study and intra and inter-species variability, resulting in an ADI of .053 mg/kg/day (Environ, 1986).

C. Pharmacokinetics

Naphthalene is rapidly absorbed when inhaled but is more slowly absorbed by ingestion or through the intact skin. No quantitative data on absorption were located. The compound is metabolized (primarily in the liver) to more toxic agents. This occurs rapidly in the adult but very slowly in the newborn (Sittig 1985). No data were located indicating naphthalene to be an hepatic enzyme inducer.

D. Environmental Effects

A variety of aquatic species has been exposed to naphthalene and most acute tests were under static procedures with unmeasured test concentrations. All but two LC50 effect levels for fish and invertebrate species are in the range of 2,300 to 8,900 ug/l. One embryo-larval test with the fathead minnow demonstrated adverse effects at a test concentration of 850 ug/l.

Daphnia magna is the only tested freshwater invertebrate species for which the acute toxicity of naphthalene has been determined (U.S. EPA, 1978) and the 48-hour EC50 is 8,570 ug/l.

DeGraeve et al. (1980) conducted flow-through tests with measured concentrations for the rainbow trout and the fathead minnow. The trout appeared to be a little more sensitive witha 96-hour LC50 of 2,300 ug/l. The 96-hour LC50 for the fathead minnow tested at 14 degrees centegrade was 4,900 ug/l and at 24 degrees centegrade the LC50 was 8,900 ug/l. The LC50 of 150,000 ug/l for the mosquitofish appears to be atypical but the result cannot be discounted.

LC50 (96 h) values for the polycheate, Neanthes arenaceodentata, Pacific oyster, and the grassshrimp are 3,800, 199,000 and 2,350 ug/l, respectively . The 24-hour LC50 values for one fish and two saltwater shrimp species range from 2,400 to 2,600 ug/l.

With the exception of the mosquitofish and the Pacific oyster, all LC50 and EC50 values, regardless of test method, fall within the narrow range of 2,300 to 8,900 ug/l for 9 freshwater and saltwater species.

Tests have been conducted to determine the chronic toxicity of naphthalene. An embryo-larval test has been conducted with the fathead minnow and the resultant chronic value is 620 ug/l. When this concentration is divided by the geometric mean LC50 value of 6,600 ug/l for this species an acute-chronic ratio of 11 is obtained. No other species have been tested under chronic conditions.

There is only one reported test that determined an apparent equilibrium bioconcentration factor for naphthalene. After nine days, the bioconcentration factor for a copepod was 5,000 . Bioconcentration data for other species for exposures of one hour to one day range from 32 to 77 and indicate that equilibrium does not occur rapidly when those results are compared to the nine-day value of 5,000.

E. Reported Levels

Naphthalene in sediments (mg/kg):

Georges Bank	.0089	Boehm and Farrington 1984
Boston Harbor	.1727	Shiaris and Jambard-Sweet 1986
Boston Harbor	.2000	Mass DEQE 1985
New York Bight	.4628	Reid et al. 1982
Long Island Sound	.9120	Reid et al. 1982
Chelsea River, MA	5.0820	Shiaris and Jambard-Sweet 198
Boston Harbor Aquarium/Fort Point	43.6280	Shiaris and Jambard-Sweet 198
Island End R. Boston MA	115.1400	unpublished data

Naphthalenes in air (ng/m3):

urban Rhode Island	0.052-0.350	Krstulovic et al. 1977

F. National Guidelines and Standards

Water Quality: none for human health; acute and chronic toxicity to aquatic life occurs at concentrations as low as 2300 ug/l and 620 ug/l respectively and would occur at lower concentrations among species more sensitive than those tested; acute toxicity to marine life occurs at concentrations as low as 2350 ug/l.

OSHA Standard: 50 mg/m3 TWA

ACGIH Threshold Limit Values: 50 mg/m3 TWA; 75 mg/m3 STEL

EPA report concerning "acceptable" soil levels derived a value of 138 mg/kg for health effects and 10 mg/kg for ecological effects (Cleland and Kingsbury, 1977); these values are not generally used by EPA today.

G. Selected State Guidelines

New York has set a water quality standard of 10 ug/l for aesthetic (taste/odour) reasons. New York also has an Interim Acceptable Ambient Level (AAL) for naphthalene in air of 166.7 ug/m3; this is derived from the ACGIH TWA-TLV.

California has developed Applied Action Levels (AALs) for water and air and is developing one for soils. The AAL for water is 18 ug/l and for air 1.8 ug/m3. These levels are applied in conjunction with the California Site Mitigation Decision Tree.

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FLUORENE

A. Physical and Chemical Properties

Property (a)	Value	Reference
Molecular weight	166.73 g/mol	Verschueren 1983
Water Solubility	1.9 ppm at 25°C	Verschueren 1983
Vapor pressure	1 to 10 x 10^{-3} mm Hg at 20-25°C	USEPA 1980
Koc	8.13 X 10^{3}	ERT 1984
Kow	1.5 X 10^{4}	Mabey et al. 1982
Kh	negligible	Mabey et al. 1982
Hc	6.4 x 10^{-5} atm-m^{3} /mole	Mabey et al. 1982

(a) Koc = organic carbon-water partition coefficient;
Kow = octanol-water partition coefficient;
Kh = hydrolysis rate constant;
Hc = Henry's Law constant

B. Toxic Effects to Humans

There are no data on the toxicity of fluorene to man (IARC 1983). IARC (1983) could not identify any studies to evaluate the carconogenicity of fluorene in man and concluded that the available data in experimental animals was inadequate to permit an evaluation of the carcinogenicity of fluorene. The U.S. Environmental \protection Agency's Carcinogen Assessment Group has not extimated a unit cancer risk (UCR) for fluorene (USEPA 1985). There are no adequate studies to estimate either a UCR or Acceptable Intake Chronic value (AIC) for flluorene.

C. Pharmacokinetics

There are no pharmacokinetic data for fluorene in man (USEPA 1980). In general, many polycyclic aromatic hydrocarbons (PAHs) can produce toxicity after inhalation, oral, or dermal exposure. Thus, it is believed that they are readily absorbed after exposure by these routes. And because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed function oxidase system. LaVoie et al. (1981 as

cited in IARC 1983) detected the fluorene metabolites 1-hydroxy-, 9-hydroxy-,and 9-ketofluorene after incubation of fluorene with rat-liver preparations. Elimination of PAHs is primarily via the hepatobiliary tract.

D. Environmental Effects

The general enviromental effects of PAH's are disscussed in the profile for Benzo(a)pyrene. There is essentilly no information on the specific environemtal effects of fluorene. However, available information in "Particulate Polycyclic Organic Matter" (NAS, 1972) suggests that fluoreneis not carcinogenic and therefore will probably not be as important as other PAH's with regard to these kinds of effects on aquatic and terrestrial animals.

E. Reported Levels

in sediments mg/kg		
Burdekin R./G B reef	.0079	Smith et al 1985
Ross R./G B reef	.0580	Smith et al 1985
New York Bight	.2569	Reid et al. 1982
USA/general	.4350	in Smith et al 1985
Townsville H./Queensland	.4646	Smith et al 1985
Long Island Sound	1.0030	Reid et al. 1982
Australia/general	1.4000	in Smith et al(1985)
Island End R. Boston MA	40	unpublished data

F. National Guidelines and Standards

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ANTHRACENE
(Paranaphthalene)

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	178.2	Mabey et al. 1982
Water Solubility	1.29 ppm at 25°C 4.5×10^{-2} ppm at 25°C	Verschueren 1983 Mabey et al. 1982
Vapor Pressure	1.7×10^{-5} Torr at 20°C	Mabey et al. 1982
Koc	1.4 x 1E + 4	Mabey et al. 1982
Kow	2.8 x 1E + 4	Mabey et al. 1982
Kh	negligible	Mabey et al. 1982
Hc	8.6×10^{-5} atm^{-m3}/mol @ 25°C	Mabey et al. 1982

(a) Koc = Organic carbon-water partition coefficient;
Kow = Octanol-water partition coefficient;
Kh = Hydrolysis rate constant;
Hc = Henry's law constant;

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult the different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

No epidemiological studies were identified which examined possible human health effects resulting from exposure to anthracene. Few reports of health effects in humans resulting from anthracene exposure exist. It is reported that three cases of epithelioma (any tumor derived from epithelium) of the hand, cheek and wrist occurred in men handling crude anthracene in an alizarin factory (Kennaway 1924 as cited in IARC 1983). In

another instance it was reported that in studies on the treatment of psoriasis, anthracene solubilized in an alcohol N-methyl-2-pyrrolidone vehicle, induced photosensitive reactions when administered topically in low concentrations (~0.25%) to humans in combination with UV radiation (Urbanek 1980, Walter 1980 as cited in IARC 1983).

Anthracene has been tested for carcinogenicity in a number of different species, using a variety of routes of administration, with primarily negative results. There is no evidence that anthracene is active in short-term tests. IARC (1983) concludes that the available data provide no evidence that anthracene is carcinogenic to experimental animals. Because of the paucity of toxicity data on anthracene, an ADI of 5.57 x 10^{-4} mg/kg/day was calculated based on the LD^{-50} value for naphthalene, another non-carcinogeneic polyaromatic hydrocarbon. Using this ADI, target concentrations of 0.019 mg/L and 0.0019 mg/m^3 were calculated for water and air.

C. Pharmacokinetics

In its review of PAHs, the U.S. EPA (1982) note that anthracene appears to be converted to 1,2-dihydroanthracene-1,2-diols and their glucuronides. In an investigation in which anthracene was incubated with rat liver preparations (Akhtar et al. 1979 as cited in IARC 1983), the major metabolite was identified as the 1,2-dihydrodiol. It has also been reported that the 1,2-dihydrodiol, 9,10-anthraquinone, 9,10-dihydrodiol, and 2,9,10-trihydroxyanthracene have been identified as metabolites in rat urine, together with conjugates consistent with the formation of the 1,2-oxide (Sims 1964 as cited in IARC 1983).

D. Environmental Effects

The profile for Benzo(a)pyrene provides a generic description of the potential environmental effects of PAH's as a class of compounds. A no effect level of 5 mg/l was observed for trout in an acute (24hour) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

E. Reported Levels

In Sediments:

	Concentration mg/kg	Reference (see Appendix A)
Penobscot Bay, ME, outer region	.0069	Johnson et al., 1985
Buzzards Bay, New Bedford, MA	.0070 - .0080	Giger and Blumer, 1974
Penobscot Bay, ME, inner region	.0234	Johnson et al., 1985
New York Bight	.0391	Reid et al., 1982
The Graves, Boston MA	.0420	Shiaris and Jambard-Sweet 1986
Boston Harbor	.0725	Shiaris and Jambard-Sweet 1986
Buzzards Bay New Bedford, MA	.1700	Giger and Blumer, 1974
Boston Harbor Aquarium/Fort Point	.2450	Shiaris and Jambard-Sweet 1986
Boston Harbor	.2833	MA DEQE, 1985
Buzzards Bay, New Bedford, MA	.3400	Giger and Blumer, 1974
Chelsea River, MA	.4110	Shiaris and Jambard-Sweet 1986
Long Island Sound	.4550	Reid et al., 1982
Savern Estuary, U.K.	2.4	John et al., 1979

In Soils:

Anthracene	0.008-0.017	U.S. EPA, 1976

In Air:

	ng/m^3	
Averages for		
residential	0.03-0.83	Radian Corp., 1983
rural	0.4	Radian Corp., 1983
urban	0.068-0.278	White & Vanderslice, 1980
urban	0.1-1.3	U.S. EPA, 1980
Detroit	1.2	White & Vanderslice, 1980

F. National Guidelines and Standards

Water quality: U.S. EPA (1980) has recommended criteria related to specific incremental lifetime risk levels of 10E-5, 10E-6, and 10E-7. The coressponding water concentrations assuming ingestion of water and aquatic organisms are 28, 2.8, and 0.28 ng/l, respectively. If the above estimates are made for consumption of aquatic organisms only, the levels are 311, 31.1, and 3.1 ng/l respectively. The values acknowledge the conservative assumption that all carcinogenic PAH's are equal in potency to Benzo(a)pyrene. The carcinogenic potency of B(a)P

and other PAH's is currently being re-evaluated by several industry groups and EPA. With regard to protectiion of aquatic life, no numerical criteria have been developed.

OSHA limit: An 8-hour TWA concentration limit of 0.2 mg/m^3 meter has been set for the benzene soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene, and pyrene).

NIOSH recommends a concentration limit for coal tar, coal tar pitch, creoscte and mixtures of these substances at 0.1 mg/m^3 meter of the cyclohexane-extractable fraction of the sample determined as a 10 hour TWA.

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PHENANTHRENE

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	178.24 g/mol	Verschueren, 1983
Water solubility	0.816 ppm @ 21°C	" "
Vapor pressure	9.6×10^{-4} torr at 25°C	" "
Koc*	1.4×10^{4}	" "
Kow	2.8×10^{4}	" "
Hc	2.26×10^{-4} atm-m^3/mol at 25°C	Lyman, Reehl, and Rosenblatt, 1982

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* Calculated from Kow using regression equation from Lyman, Reehl, and Rosenblatt (1982) for PAHs and aromatics: log Koc = log Kow - 0.21.

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under different conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

There are no data on the toxicity of phenanthrene to man (IARC, 1983). Phenanthrene has been tested for carcinogenicity in experimental animals by the oral, dermal and subcutaneous routes of administration (Huggins and Yang, 1962; Kennaway, 1924; Steiner, 1955; Grant and Roe, 1963; and Buening et al., 1979, as cited in IARC, 1983).

IARC (1983) concluded that data from available studies were inadequate to permit an evaluation of the carcinogenicity of phenanthrene in experimental animals. EPA's CAG has not estimated a CPF for phenanthrene (U.S. EPA, 1985).

C. Pharmacokinetics

There are no pharmacokinetic data for phenanthrene in (U.S. EPA, 1980). In general, many PAHs can produce toxicity after inhalation, oral or dermal exposure. Thus, it is believed that PAHs are absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed-function-oxidase system and eliminated primarily through the hepatobiliary tract.

Several metabolites of phenanthrene have been identified. They include the 1,2-, 3,4-, and 9,10-dihydrodiols, and the 1,2-diol-3,4-epoxide. The dihydrodiols displayed little or no tumor-initiating activity on mouse skin (Wood et al., 1979, as cited in IARC, 1983). The epoxide was found to be mutagenic in bacterial and mammalian cells (Wood et al., 1979, as cited in IARC, 1983). Buening and others (1979, as cited in U.S. EPA, 1985) reported significant tumorigenic activity with the epoxide, but not with phenanthrene itself in newborn Swiss mice injected intraperitoneally with 0.8/umol of either substance on days 1, 8 and 15 of life.

D. Environmental Effects

The B(a)P environmental profile discusses the general environmental effects of PAHs.

Acute toxicity of phenanthrene to fish has been reported at levels of 4,500 mg/l and would probably be lower for sensitive species or for chronic effects. There is no specific determination concerning the carcinogenic status of phenanthrene. However, available information presented in "Particulate Polycyclic Organic Matter" (NAS, 1972) suggests that phenanthrene is not carcinogenic. There is some indication that it may be mutagenic.

E. Reported Levels

In soils:	Concentration (mg/kg)	Reference (see Appendix A)
Phenanthrene	.033	U.S. EPA, 1976

In sediments:

Buzzards Bay, W. Falmouth, MA	.0330	Giger and Blumer, 1974
Penobscot Bay, ME, outer region	.0496	Johnson et al., 1985
Buzzards Bay, MA	.0530	Hites et al., 1977
The Graves/Boston Harbor	.1517	Shiaris and Jambard-Sweet, 1986
Commencement Bay, WA	.1660	Malins et al., 1982
Mystic River, Boston, MA	.4000	MA DEQE, 1985
New York Bight	1.1652	Reid et al., 1982
Boston Harbor	1.4175	MA DEQE, 1985
Boston Harbor	1.6410	Shiaris and Jambard-Sweet, 1986
Charles River, MA	3	Windsor and Hites, 1979
Long Island Sound	5.6100	Reid et al., 1982
Elliot Bay Pier 54, WA	7	Malins et al., 1982
Newton Creek, NY	14	Anderson, 1982
Chelsea River, MA	17.6577	Shiaris and Jambard-Sweet, 1986
Elizabeth River, MA	25	Malins et al., 1982
Boston Harbor Aquarium/Fort Point	32.3200	Shiaris and Jambard-Sweet, 1986
Island End River, Boston, MA	187.6500	(Unpublished data)

In air:	(ng/m^3)	
urban RI	0.11-0.340	Krstulovic et al., 1977
resididential (Florence, AL)	0.04-2.2	Radian Corp., 1983
rural (Florence, AL)	0.05-1.14	" " "

F. Guidelines and Standards

Water quality criteria: No numerical criteria have been developed for the protection of aquatic life.

OSHA limit: An 8-hour TWA concentration limit of 0.2 mg/m^3 has been set up for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene and pyrene).

NIOSH recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample determined as a 10-hour TWA.

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FLUORANTHENE

[Idryl; 1,2-(1,8-Naphthylene)benzene; Benzo(jk)fluorene]

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	202.30 g/mol	Mabey et al., 1982
Water solubility	0.265 ppm at 25°C	Verschueren, 1983
Vapor pressure	5.0×10^{-6} torr	Mabey et al., 1982
Koc	3.8×10^{4}	" " "
Kow	7.9×10^{4}	" " "
Hc	6.5×10^{-6} atm-m^3/mol	" " "

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

Fluoranthene was tested for carcinogenicity, with negative results, in several tests, including skin painting studies (Wynder and Hoffman, 1959; Hoffman et al., 1972; Horton and Christian, 1974; and Van Duuren and Goldschmidt, 1976, as cited in IARC, 1983) and a subcutaneous injection study (Shear, 1938, as cited in IARC, 1983).

Cocarcinogenic effects were evident when fluoranthene and B(a)P were applied concurrently to the skin of female mice. Groups of 50 female Swiss mice received lifetime exposures consisting of three applications a week of 40 ug fluoranthene, 40 ug fluoranthene plus 5 ug B(a)P, or 5 ug B(a)P in acetone. A vehicle control group and untreated control group were included. No tumors appeared in the fluoranthene-treated or control groups. Skin tumor incidences were 16/50 and 39/50 in B(a)P-treated and the B(a)P/fluoranthene-treated groups, respectively. Squamous-cell carcinomas constituted 12 of the 16 tumors and 37

of the 39 tumors in the B(a)P-treated and B(a)P/fluoranthene-treated groups, respectively (Van Duuren and Goldschmidt, 1976, as cited in IARC, 1983).

Conflicting evidence for the mutagenicity of fluoranthene in Salmonella typhimurium (Ames test) has been reported. Mutagenicity was found in S. typhimurium strains TA98, and TA100 and TM677, with an Aroclor-induced postmitochondrial supernatant activation system (Hermann et al., 1980; LaVoie et al., 1979; and Kaden et al., 1979, as cited in IARC, 1983). Mutations were evident in human lymphoblastoid cells with the same postmitochondrial activation system. Increased mutations due to fluoranthene were not seen in S. typhimurium strains TA1535, TA1537, TA1538, TA98 and TA100, with a similar postmitochondrial activation system (Salamone et al., 1979, as cited in IARC, 1983).

The oral LD_{50} is 2,000 mg/kg body weight (Smyth et al., 1962, as cited in IARC, 1983).

No information is available regarding other toxicity end-points, including reproductive effects and teratogenicity.

C. Pharmacokinetics

La Voie and co-workers (1982, as cited in IARC, 1983) detected the 2,3-dihydrodiol metabolite of fluoranthene. The 2,3-dihydrodiol is mutagenic in bacterial tests with an exogenous activation system. Flucranthene induced B(a)P hydroxylase in rat placental tissue (Welch et al., 1969, as cited in IARC, 1983).

No information is available regarding DNA adduct formation or intestinal and dermal absorption coefficients.

D. Reported Levels

In soils:	Concentration (mg/kg)	Reference (see Appendix A)
(unspecified)	0.11-0.79	U.S. EPA, 1976
urban	5-120	White and Vanderslice, 1980
In sediment:		
Georges Bank	.0254	Boehm and Farrington, 1984
Boston Harbor/The Graves	.0995	Shiaris and Jambard-Sweet, 1986
Penobscot Bay, ME, outer region	.1060	Johnson et al., 1985

Buzzards Bay, W. Falmouth, MA	.1100	Giger and Blumer, 1974
Buzzards Bay, MA	.1300	Hites et al., 1977
New York Bight	.2420	Reid et al., 1982
Buzzards Bay, New Bedford, MA	.7900	Giger and Blumer, 1974
Severn Estuary, UK	2.2	John et al., 1979
Penobscot Bay, ME, inner region	2.2074	Johnson et al., 1985
Long Island Sound	3.0550	Reid et al., 1982
Boston Harbor	3.8975	MA DEQE, 1985
Commencement Bay, WA	4	Malins et al., 1982
Elliot Bay Pier 54, WA	8	" " "
Newton Creek, NY	10	Anderson, 1982
Charles River, MA	13	Windsor and Hites, 1979
Mystic River, Boston, MA	19	MA DEQE, 1985
Chelsea River, MA	23.4140	Shiaris and Jambard-Sweet, 1986
Elizabeth River, VA	42	Bieri et al. in press
Boston Harbor Aquarium/Fort Point	42.8170	Shiaris and Jambard-Sweet, 1986
Island End River, Boston, MA	60.0429	(Unpublished data)
Boston Harbor	89.9714	Shiaris and Jambard-Sweet, 1986
In air:	(ng/m^3)	
U.S. cities	0.1-4.1	Fox and Staley, 1978
Lynne Center, NH	0.44	Radian Corp.. 1983
urban Hanover, NH	2.6	" " "

E. Guidelines and Standards

Water quality: U.S. EPA (1986) has established ambient water quality criteria as follows: To protect freshwater aquatic life: 3,980 ug/l based on acute toxicity test. To protect salt water life: 40 ug/l based on acute toxicity, and 16 ug/l based on chronic toxicity. To protect human health: 42 ug/l.

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PYRENE
[Benzo(def)phenanthren]

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	202.26 g/mol	Verschueren, 1983
Water solubility	0.135 ppm at 25°	ERT, 1984
Vapor pressure	6.87×10^{-7} torr at 20°C	" "
Koc	3.8×10^{4}	Mabey et al., 1982
Kow	7.6×10^{4}	ERT, 1984
Hc	5.1×10^{-6} atm^{-m^3}/mol at 25°C	Mabey et al., 1982

The values for the physical and chemical properties of various compounds are reported in the literature by diverse sources. Values differ typically because the experiments used to determine them were performed under various conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

There are no data to evaluate the toxicity of pyrene to man (IARC, 1983). Pyrene has produced negative results in most mutagenicity assays (U.S. EPA, 1982). IARC (1983) has concluded that there is limited evidence that pyrene is active in short-term mutagenicity assays.

Rats were fed pyrene at a dose of 2,000 mg of pyrene per kilogram of diet for 100 days and slightly fatty, and enlarged livers were found when the animals were sacrified. The 7- and 4-day LD_{50}s were reported to be 514 and 678 mg/kg, respectively (Salamone 1981, as cited in IARC, 1983).

IARC (1983) could not identify any studies to evaluate the carcinogenicity of pyrene in man and concluded that the available data in experimental animals provide no evidence that pyrene is carcinogenic. EPA's CAG has not estimated a CPF for pyrene (U.S. EPA, 1985).

An AIC value for pyrene can be estimated from subchronic feeding studies. Using the subchronic LOEL of 2,000 mg pyrene/kg of diet and assuming the rats weighed 250 g and consumed 15 g/food/day, an uncertainty factor of 1,000 and a severity factor of 2, the AIC is calculated to be 0.06 mg/kg/day.

C. Pharmacokinetics

There are no pharmacokinetic data for pyrene in man (U.S. EPA, 1980). In general, many PAHs can produce toxicity after inhalation, oral or dermal exposure. Thus, it is believed that PAHs are absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Pyrene, like other PAHs, is apparently metabolized by the microsomal mixed-function-oxidase system in mammals. The 1-hydroxy, 1,6-, 1,8-dihydroxy, and the 4,5-dihydrodiol have been identified as metabolites of pyrene in rats and rabbits (Boyland and Sims, 1964, as cited in IARC, 1983), and two trihydroxy derivatives were isolated by Jacob and others (1982, as cited in IARC, 1983).

The elimination of pyrene from rats exposed to a pyrene aerosol (500 mg/l, 0.3-0.5 mm particles) for 60 minutes was reported by Mitchell and Tu (1979, as cited in U.S. EPA, 1982) to occur rapidly primarily through the liver and biliary system. When 50 ug pyrene was administered in a gelatin-saline suspension to two rats by a stomach tube, approximately half of the administered pyrene was still present in the gastrointesinal tract after 24 hours (Mitchell and Tu, 1979, as cited in U.S. EPA, 1982).

D. Environmental Effects

Pyrene is expected to be similar to other PAH compounds. The environmental profile for B(a)P discusses the general environmental effects of PAH compounds.

No data were found on the toxicity of pyrene of freshwater animals. The carcinogenic status of this chemical has not been determined. However, available information presented in "Particulate Polycyclic Organic Matter" (NAS, 1972) suggests that pyrene is not carcinogenic.

E. Reported Levels

In soils:	Concentration (mg/kg)	Reference (see Appendix A)
urban	0.1-0.96	U.S. EPA, 1976
urban	5-120	White and Vanderslice, 1980
In sediments:		
Heron I./Queensland	.0013	Smith et al., 1985
Burdekin R./G B reef	.0064	" " "
Ross R./G B reef	.0440	" " "
Buzzards Bay, W. Falmouth, MA	.1000	Giger and Blumer, 1974
Buzzards Bay, MA	.1200	Hites et al., 1977
New York Bight	.2633	Reid et al., 1982
Gladstone H./Queensland	.2700	Smith et al., 1985
Japan/general	.4350	" " "
USA/general	.5300	" " "
Buzzards Bay, New Bedford, MA	.9600	Giger and Blumer, 1974
The Graves/Boston Harbor	1.2320	Shiaris and Jambard-Sweet, 1986
Townsville H./Queensland	1.2555	Smith et al., 1985
Boston Harbor	1.3071	MA DEQE, 1985
Severn Estuary, UK	1.9	John et al., 1979
Boston Harbor	2.4485	Shiaris and Jambard-Sweet, 1986
Long Island Sound	3.2850	Reid et al., 1982
Commencement Bay, WA	4	Malins et al., 1982
Australia/general	4.5000	Smith et al., 1985
Newton Creek, NY	7	Anderson, 1982
Elliot Bay Pier 54, WA	11	Malins et al., 1982
Charles River, MA	12	Windsor and Hites, 1979
Mystic River, Boston, MA	12	MA DEQE, 1985
Chelsea River, MA	21.1543	Shiaris and Jambard-Sweet, 1986
Elizabeth River, VA	28	Bieri et al. (In press)
Boston Harbor Aquarium/Fort Point	35.0130	Shiaris and Jambard-Sweet, 1986
Island End River, Boston, MA	151.3333	(Unpublished data)

In air:	(ng/m^3)	
urban	0.18-5.2	Fox and Staley, 1978; Gordon and Bryan, 1973
residential (Florence, AL)	0.12-4.2	Radian Corp, 1983
rural (Florence, AL)	0.09-0.49	" " "

F. Guidelines and Standards

OSHA limit: An 8-hour TWA concentration limit of 0.2 mg/m^3 has been set for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene and pyrene).

NIOSH recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA.

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Pyrene

ACENAPHTHALENE
[Cyclopenta(de)naphthalene]

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	152.2	Mabey et al., 1982
Water solubility	3.93 ppm at 24°C	Verschueren, 1983
Vapor pressure	2.9×10^{-2} at 20°C	Mabey et al., 1982
Koc	2.5×10^{3}	" " "
Kow	5.3×10^{3}	" " "
Hc	1.45×10^{-3} atm$^{-m^3}$/mol	" " "

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

There are no long-term studies in the literature that evaluate the carcinogenicity of acenaphthalene (Sedman, 1985) nor are there any data from epidemiologic studies which correlate acenaphthalene exposure with an increased risk of cancer. Structurally, acenaphthalene is similar to other low-molecular weight PAHs that are considered to be non-carcinogenic.

Little information regarding the acute or chronic toxicity of acenaphthalene was located. Knobloch and others (1969) reported a subchronic study in which rats were given oral doses of 0.6 g/kg acenaphthalene for 40 days or 0.3 g/kg for 32 days. In the high-dose group, the toxic effects reported included significant decreases in body weight, in some organ weights, and in the lymphocyte count. They also reported toxic effects on the liver, including fatty accumulation, degeneration and focal necrosis of the parenchyma. Kidney damage was also observed as well as heart and spleen atrophy. In the low-dose group, the authors reported

significant decreases in body and kidney weights, increased activity of the enzymes alanine and aspartate aminotransferase, an increase in urine volume, and minor signs of liver and kidney degeneration. The use of this study in developing AIC concentrations is questionable because of a number of methodological flaws, including an inadequate number of animals per dose group (7-8); the short length of the study; lack of information on the sex of the animals; and an inadequate description of methodology. The same investigators reported oral LD_{50} values in mice and rats of 2.15 g/kg and 3.0 g/kg, respectively.

The low dose of 0.3 g/kg/day reported in the Knobloch study is equivalent to a subchronic LOAEL. An uncertainty safety factor of 1,000 is used to extrapolate an animal subchronic NOEL to a human chronic NOEL. A severity factor of 10 is applied to account for observed effects and the inadequacy of the methodology, resulting in an AIC of 0.03 mg/kg/day.

Positive results have also been reported from a single mutagenicity test in which acenaphthalene was tested in a strain of Salmonella typhimurium in the presence of liver microsomal activation (U.S. EPA, 1982).

C. Pharmacokinetics

No data were located on the pharmacokinetic properties of acenaphthalene in laboratory animals or man. In the absence of data, it is assumed that 100% of acenaphthalene is absorbed through the oral or inhalation exposure routes.

D. Environmental Effects

A general review of the environmental fate and effects of PAH compounds is provided in the environmental profile on benzo(a)pyrene.

E. Reported Levels

No data obtained at this writing.

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ALDRIN/DIELDRIN

Summary

Aldrin degrades to dieldrin, which is very persistent in the environment. Both pesticides are carcinogenic in rats and mice and are teratogenic and reproductive toxicants. Aldrin and dieldrin cause liver toxicity and central nervous system abnormalities following chronic exposure. Both are also acutely toxic with oral LD50 values of about 50 mg/kg. Both pesticides are very toxic to aquatic organisms and have been associated with large-scale kills of terrestrial wildlife in treated areas.

Background Information

Dieldrin is the 6,7-epoxide of aldrin and is readily obtained from aldrin under normal environmental conditions and by metabolism in animals.

CAS Number: Aldrin: 309-00-2
Dieldrin: 60-57-1

Chemical Formula: Aldrin: $C_{12}H_8Cl_6$
Dieldrin: $C_{12}H_8Cl_6O$

IUPAC Name: Aldrin: 1,2,3,4,10,10-hexachloro-1,4a,5,8,8a-hexahydro-1,4:5,8-exo-dimethanonaphthalene

Dieldrin: 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-endo,exo-1,4:5,8-dimethanonaphthalene

Chemical and Physical Properties

Molecular Weight: Aldrin: 365
Dieldrin: 381

Melting Point: Aldrin: 104°C
Dieldrin: 176°C

Solubility in Water: Aldrin: 20 ug/liter at 25°C
Dieldrin: 200 ug/liter at 25°C

Solubility in Organics: Soluble in most organic solvents

Log Octanol/Water Partition Coefficient: No data found; probably greater than 5 for both chemicals

Vapor Pressure: Aldrin: 2.31×10^{-5} mm Hg at 20°C
Dieldrin: 2.8×10^{-6} mm Hg at 20°C

Transport and Fate

Aldrin evaporates rapidly from aquatic environments and also probably from soil. Photolysis probably occurs in the atmosphere after volatilization. Adsorption, especially by organic materials, is also an important fate process for this chemical. Aldrin is bioconcentrated by aquatic organisms by a factor of 10^3 to 10^4. Biotransformation by aquatic organisms and biodegradation are also important fate processes.

The primary product of aldrin degradation is its epoxide, dieldrin. Photolysis of aldrin also produces small amounts of photoaldrin, photodieldrin, and a polymerization product. Dieldrin is considered to be at least as toxic as aldrin and is quite persistent in the environment. Therefore, transformation of aldrin represents only a change of state and not detoxification of the chemical.

Dieldrin is one of the most persistent of the chlorinated hydrocarbons. Volatilization and possibly subsequent photolysis to photodieldrin are important transport and fate processes from surface water and probably from soil. Adsorption to sediments, especially organic materials, and bioaccumulation are also important in removing dieldrin from water. Biotransformation and biodegradation of dieldrin occur very slowly but may be the final fate processes in sediment.

Health Effects

Both aldrin and dieldrin are carcinogens, causing increases in a variety of tumors in rats at low but not at high doses and producing a higher incidence of liver tumors in mice. The reason for this reversed dose-response relationship is unclear. Neither appears to be mutagenic when tested in a number of systems. Aldrin and dieldrin are both toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation; while teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Chronic effects attributed to aldrin and dieldrin include liver toxicity and central nervous system abnormalities. Both chemicals are acutely toxic; the oral LD_{50} is around 50 mg/kg, and the dermal LD_{50} is about 100 mg/kg.

Toxicity to Wildlife and Domestic Animals

Aldrin and dieldrin are both acutely toxic to freshwater species at low concentrations. Tests in fish showed that the two chemicals had similar toxicities, with LC_{50} values ranging from 1 to 46 ug/liter for different species. Final acute values for freshwater species were determined to be 2.5 ug/liter for dieldrin and 3.0 ug/liter for aldrin. Saltwater species were also quite sensitive to aldrin and dieldrin. The range of LC_{50}

values was similar to that for freshwater species: 2 to 100 ug/liter for aldrin and 1 to 34 ug/liter for dieldrin. The saltwater Final Acute Values were 1.3 ug/liter for aldrin and 0.71 ug/liter for dieldrin.

Chronic studies have been conducted on the effects of dieldrin on freshwater and saltwater species. For freshwater organisms, chronic values as low as 0.2 ug/liter were obtained. The Final Acute Chronic Ration was determined to be 8.5, and the calculated Freshwater Final Chronic Value was 0.29 ug/liter. Only one chronic study was done on saltwater species. Therefore, the saltwater Final Chronic Value of 0.084 ug/liter was determined by dividing the Final Acute Value by the acute-chronic ration. No chronic studies were performed on aldrin, but because its acute toxicity is comparable to that of dieldrin and because it is readily converted to dieldrin in animals and in the environment, it probably has similar chronic toxicity.

Both pesticides, and especially dieldrin, have been associated with large-scale bird and mammal kills in treated areas. Experimental feeding studies have shown that the chemicals are quite toxic to terrestrial wildlife and domestic animals at low levels.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: Aldrin: 3.0 ug/liter
Dieldrin: 2.5 ug/liter

Chronic toxicity: Aldrin: No available data
Dieldrin: 0.0019 ug/liter

Saltwater

Acute toxicity: Aldrin: 1.3 ug/liter
Dieldrin: 0.71 ug/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations in water are:

Risk	Aldrin Concentration	Dieldrin Concentrations
10^{-5}	0.74 ng/liter	0.71 ng/liter
10^{-6}	0.074 ng/liter	0.071 ng/liter
10^{-7}	0.0074 ng/liter	0.0071 ng/liter

CAG United Risk (USEPA): Aldrin: 17.0 (mg/kg/day)$^{-1}$
Dieldrin: 16.0 (mg/kg/day)$^{-1}$

RfD Value Aldrin 0.00003 (mg/kg/day)
Dieldrin 0.00005 (mg/kg/day)

ACGIH Threshold Limit Value:* 0.25 mg/m^3 TWA
0.75 mg/m^3 STEL

OSHA Standard (air):* 250 ug/m^3 TWA

* Applies to both aldrin and dieldrin.

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HEPTACHLOR

Summary

Heptachlor is an organochlorine pesticide. Together with its active metabolite, heptachlor epoxide, it is very persistent in the environment. When administered orally to mice, both substances cause liver tumors. They also have mutagenic effects. These chemicals have a number of reproductive and teratogenic effects, including decreased litter size, shortened life span of suckling young, and the development of cataracts in offspring. The acute toxicity of both heptachlor and heptachlor epoxide is very high. Chronic exposure induces liver changes and may cause kidney damage. Heptachlor is also highly toxic to fish and wildlife.

Background Information

Technical heptachlor is a complex mixture containing approximately 72 % heptachlor and 28% related compounds. It is a soft wax with a melting point of 46-74°C.

CAS Number: 76-44-8

Chemical Formula: $C_{10}H_5Cl_7$

IUPAC Name: 1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene

Chemical and Physical Properties

Molecular Weight: 373.3

Melting Point: 95-96°C

Specific Gravity: 1.57-1.59 at 9°C

Solubility in Water: 0.056 to 0.180 mg/liter at 25-29°C depending on particle size

Solubility in Organics: Soluble in ethanol, ether, benzene, acetone, carbon tetrachloride, xylene, kerosene, cyclohexanone, and ligroin

Vapor Pressure: 0.0003 mm Hg at 25°C

Transport and Fate

Heptachlor and its active metabolite, heptachlor epoxide, are very persistent in the environment, resisting chemical and biological breakdown into harmless substances. Sorption of heptachlor to sediments appears to be an important process for removal of the pesticide from water, as residue concentrations in sediment are often much higher than in water. Some volatilization may also occur.

Heptachlor and heptachlor epoxide bind tightly to soil particles and will persist for years in soil after surface application. However, heptachlor applied as an emulsifiable concentrate is more readily volatized than when applied as a granular formulation. Certain crops accumulate residues of these compounds by absorption from the soil.

Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur. Heptachlor and heptachlor epoxide are widespread in ambient air, but generally occur at low concentrations. However, levels vary both geographically and seasonally.

Health Effects

Heptachlor and heptachlor epoxide are liver carcinogens when administered orally to mice. Results from mutagenicity bioassays suggest that these compounds also may have genotoxic activity. Reproductive and teratogenic effects in rats include decreased litter size, shortened life span of suckling rats, and development of cataracts in offspring.

Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects due to heptachlor exposure. Although heptachlor and heptachlor epoxide are absorbed most readily through the gastrointestinal tract, inhalation and skin contact are also potential routes of exposure. Acute exposure by various routes can cause development of hepatic vein thrombi and can affect the central nervous system and cause death. Chronic exposure induces liver changes, affects hepatic microsomal enzyme activity, and causes increased mortality in offspring. The oral LD_{50} in the rat is 40 mg/kg for heptachlor and 47 mg/kg for heptachlor epoxide.

Although there are reports of acute and chronic toxicity in humans, with symptoms including tremors, convulsions, kidney damage, respiratory collapse, and death, details of such episodes are not well documented. Heptachlor epoxide has been found in a high percentage of human adipose tissue samples, and also in human milk samples and biomagnification of heptachlor/heptachlor epoxide occurs. This compound also has been found in the tissues of stillborn infants, suggesting an ability to cross the placenta and bioaccumulate in the fetus.

Toxicity to Wildlife and Domestic Animals

Heptachlor is toxic at low concentrations in some aquatic invertebrate and fish species. Heptachlor epoxide appears to be a minor product of heptachlor transformations in aquatic systems but the capability of different organisms to effect epoxidation varies. Mean acute values for freshwater species range from 0.9 to 78 ug/liter for invertebrates and from 13.1 to 320 ug/liter for fish. A life cycle test conducted with the fathead minnow provides a chronic value of 1.26 ug/liter and an acute-chronic ratio of 80 for this species. Saltwater mean acute values range from 0.04 to 194 ug/liter for a variety of fish and invertebrate species. A chronic value of 1.58 ug/liter and an acute-chronic ratio of 3.9 are reported for the sheepshead minnow.

Heptachlor shows a strong tendency to bioaccumule. It can concentrate at levels thousands of times greater than those in the surrounding water in a variety of aquatic organisms. Because of this tendency for bioaccumulation, chronic exposure to levels greater than 0.004 ug/liter is considered potentially harmful to aquatic life. However, this value may be too high because to average concentration in a high lipid species will be at FDA action levels for human consumption.

Heptachlor and heptachlor epoxide residues have been found in a wide variety of wildlife and domestic animal species, but usually at relatively low levels.; The use of heptachlor as a seed dressing for cereal grains has been linked to mortality among granivorous birds and to increased residues have been found in a wide variety of wildlife and domestic animal species, but usually at relatively low levels. The use of heptachlor as a seed dressing for cereal grains has been linked to mortality among granivorous birds and to increased residues in the tissues of granivorous birds and mammals. Residues have also been found in raptors but a causal relationship with observed toxic effects has not been established. Increased mortality among birds, mammals, fish, and aquatic species has been reported in areas treated with heptachlor. Heptachlor or heptachlor epoxide residues have regularly been found in food and feed crops, meat, fish, poultry, dairy products, and eggs. Oral LC_{50} values for heptachlor ranging from 92 to 480 ppm in their diet (around 20 mg/kg body weight) are reported for wild bird species.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: 0.52 ug/liter
Chronic toxicity: 0.0038 ug/liter

Saltwater

Acute toxicity: 0.053 ug/liter
Chronic Toxicity: 0.0036 ug/liter

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of heptachlor in water are:

Risk	Concentration
10^{-5}	2.78 ng/liter
10^{-6}	0.28 ng/liter
10^{-7}	0.028 ng/liter

CAG Unit Risk (USEPA): 3.37 $(mg/kg/day)^{-1}$

OSHA Standard (skin): 0.5 mg/m^3 TWA

ACGIH Threshold Limit Values (skin): 0.5 mg/m^3 TWA
2 mg/m^3 STEL

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DDT

Summary

DDT is an organochlorine pesticide, which together with its metabolites, is very persistent in the environment. DDT, DDE, and DDD have been shown to be carcinogenic in mice. They primarily cause liver tumors, but they also increase the incidence of lung tumors and lymphomas. In addition, DDT is a reproductive toxin. Chronic exposure can damage the central nervous system and liver. DDT and other organochlorine pesticides are highly toxic to aquatic organisms and are responsible for the decreased reproductive success of many bird species.

Background Information

Technical DDT is a mixture containing 65-80% p,p'-DDT, 15-20% o,p-DDT, up to 4% p,p'-DDD, and traces of other materials. Metabolites of DDT include p,p'-DDE and o,p'-DDD. The DDT isomers and metabolites are usually found together and generally have similar properties; therefore, they will be considered together. Where differences occur the specific isomer will be identified. DDT will be used to refer to the combination of technical material and metabolites. Specific DDT isomers will be identified as such.

CAS Number:
p,p'-DDT: 50-29-3
o,p'-DDT: 789-02-6
p,p'-DDD: 72-54-8
o,p'-DDD: 53-19-0
p,p'-DDE: 72-55-9

Chemical Formula:
p,p'- and o,p'-DDT: $C_{14}H_9Cl_5$
p,p'- and o,p'-DDD: $C_{13}H_{10}Cl_4$
p,p'- and o,p'-DDE: $C_{14}H_8Cl_4$

IUPAC Name:
p,p'-DDT: 1,1,1-Trichloro-2,2-bis(4-chlorophenyl)ethane
o,p'-DDT: 1,1,1-Trichloro-2-(2-chlorophenyl)-2-(4-chlorophenyl)ethane
p,p'-DDD: 1,1-Dichloro-2,2-bis(4-chlorophenyl)-ethane
o,p'-DDE: 1,1-Dichloro-2,2-bis(4-chlorophenyl)-ethene

Important Synonyms and Trade Names:

DDT: Dichlorodiphenyltrichloroethane, dicophane, chlorophenothane, Gesarol, Neocid
p,p'-DDD: TDE, Rothane

Chemical and Physical Properties

Molecular Weight: o,p′- and p,p′-DDT: 354.5
DDD: 320
DDE: 318

Boiling Point: DDT: 260°C

Melting Point: DDT: 109°C
DDD: 112°C
DDE: 90°C

Solubility in Water: p,p′-DDT: 5.5 ug/liter
o,p′-DDT: 26 ug/liter
p,p′-DDD: 20 ug/liter
DDE: 14 ug/liter

Solubility in Organics: DDT: Soluble in acetone, benzene, cyclohexanane, morpholine, pyridine, and dioxane

Log Octanol/Water Partition Coefficient:

DDT: 4.98
p,p′-DDT: 3,98
p,p′-DDD: 5.99
o,p′-DDD: 6,08
DDE: 5.69

Vapor Pressure:

p,p′-DDT: $1.9\text{x}10^{-7}$ mm Hg at 25°C
p,p′-DDT: $7.3\text{x}10^{-7}$ mm Hg at 30°C
o,p′-DDT: $5.5\text{x}10^{-6}$ mm Hg at 30°C
p,p′-DDD: $1.0\text{x}10^{-6}$ mm Hg at 30°C
o,p′-DDD: $1.9\text{x}10^{-6}$ mm Hg at 30°C
DDE: $6.5\text{x}10^{-6}$ mm Hg at 20°C

Transport and Fate

DDT and its metabolites are very persistent in the environment. Volatilization is probably the most important transport process from soil and water for p,p′-DDT and o,p′-DDT, as evidenced by the ubiquitous nature of DDT in the environment. Sorption and bioaccumulation are the most important transport processes for the DDT isomers. Although it only occurs slowly, the ultimate fate process for p,p′-DDT, o,p′-DDT, and DDD is biotransformation to form bis(2-chlorophenyl)methanone (DDCO). Indirect photolysis may also be important for p,p′-DDT and o,p′-DDT in aquatic environments. For DDE, direct photolysis is the most important ultimate fate process in the environment although biotransformation may also be important.

Health Effects

DDT, DDE, and DDD have been shown to be carcinogenic to mice, primarily causing liver tumors, but also causing lung tumors and lymphomas. DDT does not appear to be mutagenic, but it has caused chromosomal damage. There is no evidence that DDT is a teratogen; but it is a reproductive toxin, causing reduced fertility, reduced growth of offspring, and fetal mortality.

Chronic exposure to DDT causes a number of adverse effects, especially to the liver and central nervous system (CNS). DDT induces various microsomal enzymes and therefore probably affects the metabolism of steroid hormones and exogenous chemicals. Other effects on the liver include hypertrophy of the parenchymal cells and increased fat deposition. In the CNS, exposure to DDT causes behavioral effects such as decreased aggression and decreased conditional reflexes. Acute exposure to large doses or chronic exposure to lower doses causes seizures. The oral LD_{50} is between 113 and 450 mg/kg for the rat and is generally higher for other animals.

DDT, DDD, and DDE are bioconcentrated and stored in the adipose tissues of most animals.

Toxicity to Wildlife and Domestic Animals

DDT has been extensively studied in freshwater invertebrates and fishes and is quite toxic to most species. The range of toxicities was 0.18 to 1,800 ug/liter and the freshwater final acute value for DDT and its isomers was determined by EPA to be 1.1 ug/liter. Saltwater species were somewhat more sensitive to DDT; the saltwater final acute value for the DDT isomers was 0.13 ug/liter. Only one chronic toxicity test on aquatic species was reported. This test indicated that the acute-chronic ratio for DDT might be high (65 in the reported study), but the data were insufficient to allow calculation of a final acute-chronic ratio. DDT,DDD, and DDE are bioconcentrated by a factor of 10^3 to 10^5.

DDT, DDD, DDE and the other persistent organochlorine pesticides are primarily responsible for ;the great decrease in the reproductive capabilities and consequently in the populations of fish-eating birds, such as the bald eagle, brown pelican, and osprey. DDT has also been shown to decrease the populations of numerous other species of waterbirds, raptors, and passerines significantly.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

DDT: Freshwater

Acute toxicity: 1.1 ug/liter

Chronic toxicity: 0.001 ug/liter

Saltwater

Acute toxicity: 0.13 ug/liter
Chronic toxicity: 0.001 ug/liter

DDD and DDE: The available data are not adequate for establishing criteria. However, EPA did report the lowest values known to be toxic in aquatic organisms.

Freshwater

Acute toxicity: DDD: 0.6 ug/liter
DDE: 1050 ug/liter
Chronic toxicity: DDD & DDE: No available data

Saltwater

Acute toxicity: DDD: 3.6 ug/liter
DDE: 14 ug/liter
Chronic toxicity: DDD & DDE: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of DDT in water are:

Risk	Concentration
10^{-5}	0.24 ng/liter
10^{-6}	0.024 ng/liter
10^{-7}	0.0024 ng/liter

CAG Unit Risk (USEPA): 0.34 (mg/kg/day)-1

OSHA Standard (air): 1mg/m^3 TWA

ACGIH Threshold Limit Value: 1 mg/m^3 TWA

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HEXACHLOROCYCLOHEXANE

Summary

Hexachlorocyclohexane (HCH) has four major isomers, alpha, beta, gamma, and delta, of which the gamma isomer (lindane) is generally the most active. HCH is fairly persistent in the environment. Three of the isomers caused liver tumors in mice when administered alone. Exposure to lindane decreased the number of live young produced by pregnant dogs. Lindane is also quite toxic to aquatic life.

Note: Information presented below should be considered generally applicable to the HCH isomers unless a specific isomer is indicated.

CAS Number: 608-73-1
alpha-HCH: 319-84-6
beta-HCH: 319-85-7
gamma-HCH: 58-89-9
delta-HCH: 319-86-8

Chemical Formula: $C_6H_6Cl_6$

IUPAC Name: 1,2,3,4,5,6-Hexachlorocyclohexane

Important Synonyms and Trade Names: Benzene hexachloride, HCH, Lindane (gamma-HCH), HCH

Chemical and Physical Properties

Molecular Weight: 290.82

Boiling Point: No available data

Melting Point:
alpha-HCH: 158°C
beta-HCH: 310°C
gamma-HCH: 112°C
delta-HCH: 138°C
Technical HCH: 65°C

Solubility in Water:
alpha-HCH: 10mg/liter
beta-HCH: 5 mg/liter
gamma-HCH: 10 mg/liter
delta-HCH: 10 mg/liter
Technical HCH: 10-32 mg/liter

Log Octanol/Water Partition Coefficient: 3.8

Vapor Pressure:
alpha-HCH: 2.5×10^{-5} mm Hg at 20°C
beta-HCH: 2.8×10^{-7} mm Hg at 20°C
gamma-HCH: 2×10^{-4} mm Hg at 20°C

delta-HCH: 1.7×10^{-5} mm Hg at 20°C

Transport and Fate

In general, the transport and fate of the hexachlorocyclohexane isomers is similar and they will therefore be discussed as a group. The primary transport and fate process for hexachlorocyclohexane in an aqueous system appears to be adsorption to organic particles, transport to anaerobic sediments, and subsequent biodegradation by anaerobic organisms. Volatilization may be somewhat important in the aquatic environment and is probably a major transport process in soils. It is important to note that biodegradation of hexachlorocyclohexane yields such chemicals as pentachlorocyclohexane, tetrachlorobenzene, and trichlorophenol and therefore may not result in substantial detoxification of the chemical. Lindane has been shown to be rather persistent when applied to soil, with up to 10 percent of an applied sample remaining after 10 years.

Health Effects

The alpha, beta, and gamma isomers of hexachlorocyclohexane have all been shown to cause liver tumors in mice but not in other tested species. HCH has not been thoroughly tested for genotoxic effects but does not appear to be mutagenic. ; The alpha, beta, and delta isomers have not been tested for their teratogenic or reproductive toxicological potential. Lindane has been tested and was not teratogenic, but in two studies it decreased the number of live young produced (Earl et al. 1973). Alpha-HCH has been shown to cause nonmalignant lesions in the liver of test animals at doses below those required to induce tumors. Lindane has been associated with the development of aplastic anemia in humans (West 1967).

Toxicity to Wildlife and Domestic Animals

Lindane (gamma-HCH) is responsible for the effectiveness of hexachlorocyclohexane as an insecticide and is generally more toxic than the other isomers or technical HCH. In fact, the presence of the other HCH isomers decreases the toxicity of lindane to aquatic organisms, either by an antagonistic effect or by decreasing the chemical's solubility. Therefore, the toxicity of lindane and HCH will be considered separately.

Lindane is acutely toxic to freshwater fish with LC_{50} water fish at levels of from 7.3 to 104 ug/liter. Lindane was acutely toxic to the pink shrimp at 0.17 ug/liter. Acute-chronic ratios for lindane ranged from 7.5 to 63, and therefore the Final Chronic Value for the protection of freshwater species was determined to be 0.08 ug/liter. Aquatic organisms appear to bioconcentrate between 100 and 500 times the steady-state concentration of lindane in the water.

Technical hexachlorocyclohexane was much less toxic than lindane, with acute toxicity ranging from 100 ug/liter to 15,000 ug/liter for freshwater fish/ Data on saltwater species also indicated that the technical compound was less acutely toxic. No information was available on the chronic toxicity of HCH. A bioconcentration factor was not reported but is probably similar to that for lindane.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Hexachlorocyclohexane mixture:

The available data are inadequate for establishing final criteria for hexachlorocyclohexane mixture. However, EPA did report the lowest values known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity: 100 ug/liter
Chronic toxicity: No available data

Saltwater

Acute toxicity: 0.34 ug/liter
Chronic toxicity: No available data

Lindane (gamma-HCH):

Freshwater

Acute toxicity: 2.0 ug/liter
Chronic toxicity: 0.08 ug/liter

Saltwater

Acute toxicity: 0.16 ug/liter
Chronic toxicity: No available data

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of the HCH isomers in water are:

Risk	Alpha-HCH Concentration	Beta-HCH Concentration	Gamma-HCH Concentraton	Technical HCH Concentration
10^{-5}	92 ng/liter	163 ng/liter	186 ng/liter	123 ng/liter
10^{-6}	9.2 ng/liter	16.3 ng/liter	18.6 ng/liter	12.3 ng/liter

10^{-7}	0.92 ng/liter	1.63 ng/liter	1.86 ng/liter	1.23 ng/liter

Interim Primary Drinking Water Regulation: gamma-HCH: 0.004 mg/liter

CAG Unit Risk (USEPA):
alpha-HCH: 6.30 $(mg/kg/day)^{-1}$
beta-HCH: 1.80 $(mg/kg/day)^{-1}$
gamma-HCH: 1.30 $(mg/kg/day)^{-1}$
technical-HCH: 4.75 $(mg/kg/day)^{-1}$

OSHA Standard (air):?: gamma-HCH: 500 ug/m^3 TWA

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BENZENE

Information presented below has been assembled to aid in the preparation of risk assessments. Detailed data on health end environmental effects can be found in the bibliography provided at the end of this profile.

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	78.12 g/mol	Verschueren 1983
Water solubility	1.78 x 103 ppm at 25°C	Mabey et al. 1982
Vapor pressure	95.2 mm Hg at 25°C	Mabey et al. 1982
Koc	6.5 x 10E1	Mabey et al. 1982
Kow	1.35 x 10E2	Mabey et al. 1982
Kh	negligible	Mabey et al. 1982
Hc	5.5×10^{-3} atm-m^3/mol at 25°C	Mabey et al. 1982

(a) Koc = organic carbon-water partition coefficient;
Kow = octanol-water partition coefficient;
Kh = hydrolysis rate constant;
Hc = Henry's Law constant

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

Acute exposure to benzene results in nausea, vomiting, ataxia and excitement. Benzene depresses central nervous system function. Narcotic effects, similar to those produced by toluene 'glue sniffing', may occur. Chronic exposure to benzene is known to cause pancytopenia, a reduction of erythrocytes, leukocytes and thrombocytes (U.S. EPA, 1980a, 1984).

Several epidemiological studies have associated occupational benzene exposure with an increase incidence of leukemia (USEPA 1984). Until recently, studies in animals did not conclusively support the evidence from humans that benzene is a leukemogen. For instance, IARC (1982) concluded that there is only limited evidence that benzene is carcinogenic in experimental animals. However, in a recent National Toxicology Program bioassay (NTP

1984), NTP found that "under the conditions of these studies, there was clear evidence of carcinogenicity of benzene" for F344/N rats and B6C3F1 mice of both sexes.

Based on epidemiological studies, the U.S. Environmental Protection Agency estimated unit cancer risks for benzene of 5.2 x 10^{-2} and 2.6 x 10^{-2} (mg/kg/day)$^{-1}$ for the oral and inhalation routes of exposure.

C. Pharmacokinetics

Benzene is absorbed by the inhalation, oral, and dermal routes of exposure since adverse effects are produced after exposure by these routes. Several studies have indicated that approximately 50% of the airborne benzene is absorbed after inhalation exposure (Teisinger et al. 1952, as cited in NIOSH 1974; Srbova et al. 1950; Hunter 1966; Nomiyama and Nomiyama 1974, as cited in Johnson 1979). The rate of dermal benzene absorption has been reported to be between 0.2 to 0.6 mg/cm^2/hr (Hanke et al. 1961; Franz 1984; Blank and McAuliffe 1985). No quantitative data on the extent of benzene absorption after oral exposure have been reported (USEPA 1984).

D. Environmental Effects

Acute toxicity data are available for two freshwater invertebrate species and six fish species (USEPA, 1978; Canton and Adema, 1978). The 48-hour effect concentrations ranged between 203,000 and 620,000 ug/l for the invertebrates. The 96-hour test results for the fish species range from 5,300 ug/l (rainbow trout) to 386,000 ug/l (mosquito fish). These results were based on different test procedures and, therefore, there is some uncertainty as to whether the differences in toxicity reflect species variations or differences in the methods.

A chronic test with the water flea Daphnia magna has been conducted but the results were incomplete (USEPA 1978). However, no adverse effects were noted at concentrations as high as 98,000 ug/l. Kauss and Hutchinson (1975) showed that there was a 50% reduction in the cell numbers of a freshwater plant after 48 hours at a concentration of 525,000 ug/l. No bioconcentration factor is available for benzene.

For saltwater species, acute values generally range between 10,900 ug/l and 924,000 ug/l. Studies have been conducted in which marine organisms were exposed to the water-soluble fraction (WSF) of oil and these data have been useful in evaluating the toxicity of light aromatic hydrocarbons as a group (the WSF contains mainly benzene, xylene, and toluene). Rice et al. 1979 examined the toxicity of WSF to 39 Alaskan marine species and

found that pelagic fish and shrimp were the most sensitive. In terms of total light aromatics, the 96 hour LC50 values ranged between 1,000-3,000 ug/l for these species.

It should be noted that based on studies of the effects of oil on aquatic/marine organisms, chronic or sublethal toxic effects can occur at low concentrations (e.g., on the order of a few ug/l). A review of this literature can be found in USEPA 1985. The studies reviewed do not focus on specific hydrocarbon compounds but on classes of compounds (e.g., soluble hydrocarbons). However, they do provide some insights into the potential for sublethal and chronic effects.

With regard to the fate of light aromatic hydrocarbons such as benzene, it should be noted that these chemicals are not expected to be persistent in the aquatic or marine environment. Therefore, exposures are most likely to occur under situations where there is a continual source of chemicals to the aquatic system. Long-term accumulation of these chemicals in sediments would not be expected nor would bioaccumulation or biomagnification in aquatic organisms.

E. Reported Levels

in air (ng/m^3):

TEAM STUDY RESULTS

Locations	Benzene Concentration (ug/m^3) Indoor Personal Air	Outdoor Air
Elizabeth/Bayone NJ		
Fall	28	9.1
Los Angeles CA		
February	18	16
May	9.2	3.6
Contra Costa CA		
June	7.5	1.9

Outdoor Air

St. Louis, MO Avg.	4,489	Singh et al. 1983
Oakland, CA Avg	4,935	Singh et al. 1983
Santa Clara County, CA	up to 6000	USEPA 1986
N. New Jersey Avg.	6,990	Hunt et al. 1984
Remote and rural sites avg.	7,520	Hunt et al. 1984
Chicago, IL AVG.	8,153	Singh et al. 1983
Philadelphia, PA Avg.	9,490	Hunt et al. 1984
Baltimore, MD Avg.	11,000	Hunt et al. 1984
Riverside, CA Avg.	12,576	Singh et al. 1983
Staten Island, NY Avg.	13,384	Singh et al. 1983
Denver, CO Avg	13,976	Singh et al. 1983
Phoenix, AZ Avg.	15,091	Singh et al. 1983
Pittsburgh, PA Avg.	15,928	Singh et al. 1983
Houston, TX Avg	18,402	Singh et al. 1983

Los Angeles, CA Avg.	19,229	Singh et al. 1983

Note: to convert from ng/m^3 to ppt divide the numbers given above by 3.2

in drinking water (ug/l):
Four of ten water supplies surveyed by EPA contained benzene at levels between 0.1-0.3 ug/l. The highest concentration reported in finished water was 10 ug/l (NAS, 1977).

F. National Guidelines and Standards

Water Quality: risk estimates have been developed for benzene in water as follows: 10E-5 (6.6 ug/l), 10E-6 (0.66 ug/l), and 10E-7 (0.066 ug/l). Acute toxicity has been observed with freshwater animals at concentrations as low as 5,300 ug/l and with saltwater organisms at concentrations as low as 5,100 ug/l. The discussion under environmental effects should be reviewed for information on possible chronic effects.

A PMCL of 5 ug/l has been proposed for benzene in drinking water.

OSHA Standards:	30 mg/m^3 TWA
	75 mg/m^3 ceiling limit
	150 mg/m^3 for 10 minute peak level

G. Selected State Guidelines

California has Applied Action Levels (AALs) for benzene of 0.2 ug/l in water and 3.2 ug/m^3 in air.

Florida has a drinking water standard of 1 ug/l for benzene.

New York has a "no detection" standard for benzene in groundwater. The state also has a guidance level of 1 ug/l in potable surface waters.

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DICHLOROMETHANE
(ALSO CALLED METHYLENE CHLORIDE)

INTRODUCTION

Methylene chloride (dichloromethane) is a solvent that is widely used for a variety of purposes. Approximately 85 percent of the methylene chloride consumed in the United States is lost directly to the environment through dispersive use, largely by evaporation to the atmosphere.

PHARMACOKINETICS

The absorption of ingested methylene chloride is virtually complete. The amount of airborne methylene chloride absorbed increases in direct proportion to its concentration in inspired air, the duration of exposure, and physical activity. Dermal absorption has not been accurately measured. Because of methylene chloride's high solubility in water and lipids, it is probably distributed throughout all body fluids and tissues. It readily crosses the blood-brain barrier. Methylene chloride is metabolized by the liver to carbon monoxide or to formaldehyde and formic acid which are further metabolized to carbon dioxide. The carbon monoxide dissociates at the lung and is eliminated (EPA, 1985a).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Acute human exposure to methylene chloride may result in irritation of eyes, skin, and respiratory tract; central nervous system depression; elevated carboxyhemoglobin levels; and circulatory disorders that may be fatal. Chronic exposure to animals may produce renal and hepatic toxicity. There is limited evidence of genotoicity in rodents (EPA, 1980).

Methylene chloride has been found positive in the Ames assay without the need for metabolic activation. Results in other tests of genotoxicity were largely negative; those for transformation were mixed. An in vivo screening test for carcinogenicity, induction of lung adenomas in strain A mice, gave suggestive positive results for methylene chloride (EPA, 1980). There have been several chronic studies in which methylene chloride was administered to experimental animals either orally or by inhalation. There is clear evidence for carcinogenicity in the inhalation studies. However, there is only suggestive evidence of a treatment-associated increase in combined hepatocellular carcinomas and neoplastic nodules provided in drinking water studies (EPA, 1985a,b).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying their criteria for evaluating the overall weight of evidence of carcinogenicity, EPA has classified methylene chloride in Group B2--Probable Human Carcinogen. It was concluded that the induction of distant site tumors from inhalation exposure and the borderline significance for induction of tumors in a drinking water study are an adequate basis for concluding that methylene chloride be considered a probable human carcinogen via ingestion as well as inhalation. In its addendum to the health assessment document for dichloromethane (methylene chloride), EPA (1985b) reported carcinogenic potencies (q_1*) for exposure by inhalation and ingestion to this compound.

The q_1* for exposure by inhalation is based on the results of a National Toxicology Program (NTP) inhalation bioassay (NPT, 1985). In the NTP bioassay, groups of 50 male and 50 female F344/N rats and $B3C6F_1$ mice were exposed by inhalation 6 hours a day, 5 days a week to dichloromethane (methylene chloride) for 102 weeks. The rats were exposed to 0, 1000, or 4000 ppm. Significant increases with dose in the incidence of neoplastic lesions were reported for mammary tumors in male and female rats and for lung and liver tumors in male and female mice. The data set indicating the greatest carcinogenic potency of dichloromethane was for alveolar/ bronchiolar or hepatocellular adenomas or carcinomas in female mice, for which the incidences were 5/50, 36/48, and 46/47 for the 0-, 2000, and 4000 ppm groups, respectively. Using the molecular weight of dichloromethane (84.94g), the concentrations in ppm can be equated to concentrations in mg/liter of air ($3.5 \text{ mg/m}^3 = 1$ ppm). Assuming that the mice weighed 32 g on average, had a respiratory volume of 0.0407 m^3/day, and completely absorbed the substance, the above dosing schedule would give average daily doses of 1,582 and 3,164 mg/kg/day for the 2000- and 4000- ppm groups, respectively. These doses and incidence rates were fitted to the multistate model using the program CLOBAL83 (Howe, 1983). After using a correction factor for the difference in the surface area-to-volume ratios between mice and humans, the upper 95th percentile estimate of the carcinogenic potency (q_1*) was $1.4 \times 10^{-2} (\text{mg/kg/day})^{-1}$.

EPA (1985b) calculated a q_1* for exposure to methylene chloride by ingestion based on the results of the NTP (1985) inhalation bioassay and on an ingestion bioassay conducted by the National Coffee Association (NCA 1983). Both of these studies have limitations for estimating the risks from ingestion of this compound.

Because methylene chloride is rapidly absorbed and systematically distributed following either ingestion or inhalation, use of an inhalation study is one approach for assessing hazards from ingestion exposure. However, exposures via the two routes are likely to lead to different doses reaching individual organs. In

particular, an inhalation study may result in a higher degree of exposure to lung tissue and a lesser exposure to tissues in the digestive system. Consequently, NTP findings for liver tumors, but not lung tumors, in mice were used for quantitative estimation of risk (EPA, 1985b).

The NCA drinking water study in mice yielded suggestive but not conclusive evidence of a treatment-related increase in hepatocellular carcinoma and/or adenomas in males. EPA (1985b) used these data to make an upper-bound risk estimate from ingestion exposure. However, it was noted that because the NCA study used doses that were well below the maximum tolerated dose (MTD), elevated tumor incidences may have been found in other tissues if the study had been conducted nearer to the MTD.

The carcinogenic potency factor derived from results of the NTP study is based on the incidence of hepatocellular adenomas or carcinomas in female mice (3/50, 16/48, 40/48 in the control, 2000 ppm, and 4000 ppm groups, respectively). Using the appropriate dose-conversion procedures, a q_1* of 2.6 x 10^{-3} $(mg/kg/day)^{-1}$ was calculated.

In the NCA (1983) study, groups of from 50 to 200 B6c3F$_1$ mice received methylene chloride in drinking water for 24 months. The actual mean daily consumption levels for males and females were similar to the target levels of 60, 125, 185, and 250 mg/kg/day. Two control groups for each sex also were maintained. Significant increases in the incidence of hepatocellular adenomas and/or carcinomas were reported for male mice in the 125 mg/kg/day and 185 mg/kg/day groups, and a borderline significant increase in incidence of these neoplastic lesions was reported in the 250 mg/kg/day group. Using the incidence of hepatocellular adenomas and carcinomas in male mice (24/125, 51/200, 30/100, 31/99, and 35/125 for the control, 60-, 125-, 185-, and 250-mg/kg/day groups, respectively) and the appropriate dose-conversion procedure, a q_1* of $1.21x10^{-2}$ $(ng/kg/day)^{-1}$ was calculated.

The carcinogenic potencies calculated on the basis of the two mouse studies (NCA 1983, NTP 1985) are comparable. Consequently, EPA recommended use of the mean value of $7.5x10^{-3}$ $(mg/kg/day)^{-1}$ as the q_1* estimate for oral exposure to methylene chloride.

A risk reference dose (RfD) of 0.06 mg/kg/day for noncarcinogenic effects of methylene chloride following oral exposure has been developed by EPA (1986). The RfD was derived from a 2-year rat drinking water bioassay (NCA 1983) that identified no-observed-effect levels (NOELS) of 5.85 and 6.47 mg/kg/day for male and female rats, respectively. Liver toxicity was the observed effect, and lowest-observed-effect levels (LOELS) of 52.58 and 58.32 mg/kg/day were reported for males and females, respectively. The study was given a high confidence rating by

EPA (1986) because a large number of animals of both sexes in four dose groups were tested. Many effects were monitored and a good dose-severity relationship was observed.

In establishing an ambient water quality criterion for methylene chloride, EPA (1980) considered the end point of carboxyhemaglobin elevation for which the American Conference of Governmental Industrial Hygienists (ACGIH) had set a threshold unit value of 200 ppm. This corresponds to ingestion of drinking water from a source containing 12.4 mg/liter methylene chloride.

However, because the findings for mutagenicity and carcinogenicity were suggestive but inadequate for quantitative risk assessment, EPA adopted for methylene chloride the criterion of 0.19 ug/liter established by CAG for the structurally related substance, chloroform. In light of the information made available since publication of this criterion, it appears that the carcinogenic potencies presented in the addendum to the health assessment document for dichloromethane (EPA, 1985b) and summarized in preceding section are more appropriate for estimating risks associated with exposure to this compound.

SUMMARY OF METHYLENE CHLORIDE CRITERIA

EPA carcinogenic classification oral and inhalation	Group B2
Oral carcinogenic potency factor (q_1*)	8.0×10^{-3} $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor (q_1*)	1.4×10^{-2} $(mg/kg/day)^{-1}$
Risk Reference Dose (for noncarcinogenic effects)	0.06 mg/kg/day
AWQC	12.4 mg/liter

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CHLOROFORM

Summary

Chloroform (trichloromethane) is often produced during the chlorination of drinking water and thus is a common drinking water contaminant. It is volatile in surface waters and is not likely to be persistent in the environment. Chloroform caused an increase in kidney epithelial tumors in rats and in hepatocellular carcinomas in mice. In addition, there is suggestive evidence from epidemiological studies that exposure to chloroform and other trihalomethanes is associated with an increased incidence of bladder tumors in humans. Other toxic effects of chloroform include central nervous system depression; eye, skin, and gastrointestinal irritation; and damage to the liver, heart, and kidney.

CAS Number: 67-66-3

Chemical Formula: CCl_3

IUPAC Name: Trichloromethane

Chemical and Physical Properties

Molecular Weight: 119.38

Boiling Point: 61.5°C

Melting Point: -63.5°C

Specific Gravity: 1.4832 at 20°C

Solubility in Water: 8,200 mg/liter at 20°C

Solubility in Organics: Soluble in acetone; miscible with alcohol, ether, benzene, and ligroin

Log Octanol/Water Partition Coefficient: 1.97

Vapor Pressure: 150.5 mm Hg at 20°C

Vapor Density: 4.12

Transport and Fate

Volatilization into the atmosphere is the major transport process for removal of chloroform from aquatic systems (USEPA 1979). Once in the troposphere, chloroform is attacked by hydroxyl radicals with the subsequent formation of phosgene ($COCl_2$) and possible chlorine oxide (ClO) radicals. Neither of these reaction products is likely to persist; phosgene is readily

hydrolyzed to hydrochloric acid and carbon dioxide. Reaction with hydroxy radicals is thought to be the primary environmental fate of chloroform. However, chloroform that remains in the troposphere may return to earth in precipitation or adsorbed on particulates, and a small amount may diffuse upward to the stratosphere where it photodissociates via interaction with ultraviolet light.

Photolysis, hydrolysis, and sorption do not appear to be significant environmental fate processes for chloroform. However, sorption processes may have some importance as a removal mechanism in groundwater and soil. The log octanol/water partition coefficient indicates that this compound may bioaccumulate under conditions of constant exposure. Studies with marine organisms provide evidence for only weak to moderate bioaccumulation. Although chloroform is somewhat lipophilic and tends to be found at higher concentrations in fatty tissues, there is no evidence for biomagnification in aquatic food chains.

Health Effects

Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence of hepatocellular carcinomas in mice. (IARC 1979, USEPA 1980). Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with an increased frequency of bladder cancer in humans. However, these results are not sufficient to establish causality. An increased incidence of fetal abnormalities was reported in offspring of pregnant rats exposed to chloroform by inhalation. Oral doses of chloroform that caused maternal toxicity produced relatively mild fetal toxicity in the form of reduced birth weights. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Toxic effects include local irritation of the skin or eyes, central nervous system depression, gastro-intestinal irritation, liver and kidney damage, cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from chloroform overdosing can occur and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis.

Exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produces liver and kidney damage in laboratory animals. The oral LD_{50} and inhalation LC_{Lo} values for the rat are 908 mg/kg and 39,000 mg/m^3 per hours, respectively (ACGIH 1980).

Toxicity to Wildlife and Domestic Animals

Limited information is available concerning the toxicity of chloroform to organisms exposed at known concentrations (USEPA 1980). Median effect concentrations for two freshwater and one invertebrate species range from 28,900 to 115,000 ug/liter. Twenty-seven day LC_{50} values of 2,030 and 1,240 ug/liter were reported for embryo-larval tests with rainbow trout in water at two levels of hardness. The only reliable result concerning the toxicity of chloroform to saltwater aquatic life is a 96-hour LC_{50} value of 81,500 ug/liter for pink shrimp.

An equilibrium bioconcentration factor of six with a tissue half-life of less than 1 day was determined for the bluegill. Although chloroform is not strongly bioaccumulated, it is thought to be widely distributed in the environment and can be detected in fish, water birds, marine mammals, and various crops.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

Estimates of the carcinogenic risks associated with lifetime exposure to various concentrations of chloroform in water are:

Risk	Concentration
10^{-5}	1.90 ug/liter
10^{-6}	0.19 ug/liter
10^{-7}	0.019 ug/liter

CAG Unit Risk (USEPA): $8.1\text{x}10\text{-}2(\text{mg/kg/day})^{-1}$

Primary Drinking Water Standard: 0.10 mg/liter (total trihalomethanes)

NIOSH Recommended Standard: 9.8 mg/m^3 1-hr Ceiling Level

OSHA Standard: 244 mg/m^3 Ceiling Level

ACGIH Threshold Limit Value: 50 mg/m^3 (suspected human carcinogen)

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TOLUENE
(Methylbenzene, Methylbenzol, Phenylmethane, Toluol)

A. Physical and Chemical Properties

Property(a)	Value	Reference
Molecular Weight	92.13 g/mol	Mabey et al., 1982
Water Solubility	534.8 ppm at 25°C	Verschueren, 1983
Vapor Pressure	28.7 mm Hg at 20°C	Verschueren, 1983
Koc	300	Mabey et.al., 1982
Kow	620	Mabey et al., 1982
Hc	6.66×10^{-3}atm-m^3/mol	Mabey et al., 1982

(a) Koc = organic carbon-water partition coefficient;
Kow = octanol-water partition coefficient;
Hc = Henry's Law constant

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigatory should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

In humans, acute exposure to high levels of toluene causes CNS depression (U.S. EPA, 1980b). Exposure to 2,050 ppm of toluene caused sudden death due to heart failure. Exposure to lower levels (100-700 ppm) produced no significant effects on heart rate (U.S. EPA, 1980a). Carpenter et. al. (1944 as cited in Sandmeyer, 1981) reported that acute exposures to 800 ppm of toluene caused respiratory irritation, nasal mucous secretion, metallic taste, and impaired balance. Exposure to 600 ppm of toluene caused lassitude, hilarity, and slight nausea and exposure to 400 ppm of tolene caused mild eye lacrimation and hilarity.

The verified risk reference dose for oral exposures to toluene is 0.3 mg/kg-day (IRIS, 1988). EPA derived this RFD using a NOAEL of 300 ppm, as determined in a 24-month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. An uncertainty factor of 100 (10 for sensitive individuals and 10 for intraspecies extrapolation) was also applied in this calculation.

Available studies that assess the carcinogenicity or mutagenicity of toluene are inadequate to make conclusions regarding its potential to cause these effects (U.S. EPA, 1980b).

C. Pharmacokinetics

Toluene is absorbed after inhalation, dermal, or oral exposure. The rate of dermal toluene absorption in human subjects was reported to be 14-23 mg/cm^2/hr. (Dutkiewicz and Tyras, 1968 as cited in U.S. EPA, 1980b). No studies evaluating toluene absorption after oral exposure have been reported (U.S. EPA, 1980b).

Toluene is distributed into the tissues of the body according to their lipid content and blood perfusion: the greater the lipid content or blood perfusion, the greater the amount of toluene deposited and retained there. The relative concentration of toluene in body tissues is as follows: adipose > bone marrow > brain, liver > lung, kidney, heart, muscle (Sato et. al., 1974 as cited in U.S. EPA, 1980b).

Toluene is metabolized by the mixed function oxidase system to benzyl alcohol, which is subsequently oxidized to benzaldehyde and benzoic acid. Benzoic acid is then conjugated to glycine to form hippuric acid (U.S. EPA, 1980b). Inducers of the mixed function oxidase system decrease toluene toxicity whereas inhibitors enhance toluene toxicity. This suggests that toxicity is due to toluene directly and not to its metabolite(s).

Toluene is eliminated primarily via urinary excretion of the metabolite, hippuric acid. After inhalation exposure to toluene, a large portion of the absorbed toluene is also exhaled (U.S. EPA, 1980b).

D. Environmental Effects

Toluene is highly volatile and only slightly soluble in seawater, approximately 379 mg/l (Sutton and Calder, 1975). The transport and persistence of toluene under environmental conditions is not well known. However it is readily transferred to the atmosphere from the water surface (MacKay and Wolkoff, 1973) and may reenter the hydrosphere in rain (Walker, 1976).

U.S. EPA (1980) summarizes the toluene exposure experiments performed with marine species. The range of LC-50s for toluene in marine invertebrates other than the Pacific oyster was 3,700 ug/l (Benville and Korn, 1977) to 56,300 ug/l for mysid shrimp (U.S. EPA, 1978). Legore (1974) measured very high values for the Pacific oyster, 1,050,000 ug/l. In the admittedly limited data base, marine fish also exhibited a wide range of sensitivities. Striped bass had a LC-50 value of 6,300 ug/l (Benville and Korn, 1977) while sheepshead minnow were very resistant with LC-50s between 277,000 and 485,000 ug/l. However fish exposed to toluene can eliminate it through their gills relatively rapidly upon exposure to uncontaminated water (Thomas and Rice, 1981).

Potera (1975) examined the relationship of the 24-hour LC-50 and the varibles, temperature, salinity, and life stage of the grass shrimp. His data indicate that the LC-50 value is not particularly sensitive to these variables.

Sheepshead minnow exhibited chronic effects on hatching and survival of embryo-larvae at 5,000 ug/l (U.S. EPA, 1978).

The response of plants to toluene was variable even within species. Skeletonema costatum, a common estuarine diatom, exhibited effects on growth or photosynthesis at 8,000 ug/l (Dunstan et. al., 1975) and at concentrations greater than 433,000 ug/l (U.S. EPA, 1978). Kelp have exhibited effects on photosynthesis at 10,000 ug/l (U.S. EPA, 1980).

Of five fresh water species tested with toluene, the cladoceran, Daphnia Magna was most resistant to any acute effects. The EC-50 and LC-50 values ranged from 12,700 ug/l to 313,000 ug/l. No chronic tests were available for freshwater species. The freshwater alga tested were relatively insensitive to toluene with EC-50 values of 245,000 or greater.

E. Reported Levels

In Outdoor Air	Concentration (ng/m3)	Reference
St. Louis	5,708	Singh et. al., 1983
Oakland	11,678	
Pittsburgh	14,750	
Chicago	17,382	
Riverside	21,780	
Denver	23,432	
Phoenix	32,407	
Staten Island	33,702	
Los Angeles	44,010	

F. Guidelines and Standards

Water Quality: U.S. EPA (1987) does not suggest water quality criteria for toluene due to the limited data base. However, they do note that the available data indicate that acute and chronic toxicity to salt water aquatic life occurs at concnetrations as low as 6,300 and 5,000 ug/l respectively. The acute toxic value in freshwater is 17,500 ug/l. In saltwater, the acute value is 6,300 ug/l and the chronic value is 5,000 ug/l.

For the protection of human health from the toxic properties of toluene ingested through water and contaminated aquatic organisms, the ambient water criterion is determined to be 14.3 mg/l.

For the protection of human health from the toxic properties of toluene ingested through contaminated aquatic organisms alone, the ambient water criterion is determined to be 424 mg/l.

Drinking Water:	MCL	2.0 mg/l
	MCLG	2.0 mg/l

The EPA one-day and ten-day Health Advisories for a 10kg child are 18 mg/l and 6 mg/l, respectively.

Air:	OSHA PEL	200 ppm as an 8- hour TWA
	ceiling	300 ppm.

The ACGIH and NIOSH recommend a TLV and REL for toluene of 100 ppm, repectively (ACGIH, 1987; CDC, 1988).

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ETHYLBENZENE
(Ethylbenzol, Phenylethane)

A. Physical and Chemical Properties

Property(a)	Value	Reference
Molecular Weight	106.12 g/mol	Verschueren, 1983
Water Solubility	1.52×10^2 ppm at 20°C	Verschueren, 1983
Vapor Pressure	7.00 mm Hg at 20°C	Verschueren, 1983
Koc	1.1×10^3 ml/g	P.H.R.E.D.
Kow	1.4×10^3	Verschueren, 1983
Hc	6.43×10^{-3} atm-m^3/mol at 20°C	P.H.R.E.D.

(a) Koc = organic carbon-water partition coefficient;
Kow = octanol-water partition coefficient;
Hc = Henry's Law constant.

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiles such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

Bardodejova and Bardodej (1970) reported no toxic effects after inhalation exposures of 100 ppm ethyl benzene for 8 hours by human volunteers. An increase in this concentration resulted in fatigue, headache, and mild eye and respiratory irritation.. The TLV for ethyl benzene is 100 ppm and was set to protect against its irritant effects. At this concentration no systemic effects of ethyl benzene are expected to be produced (ACGIH, 1980).

No data on teratogenic and reproductive activity of ethyl benzene were located by U.S. EPA (1980).

No data on the mutagenicity of ethyl benzene were located by U.S. EPA (1980). Several of its metabolites, mandelic acid, phenylglyoxylic acid, and hippuric acid gave negative results in the Ames Salmonella typhimurium mutagenicity assay (U.S. EPA, 1980).

No data concerning the carcinogenic effects of this compound were found in the literature.

The U.S. EPA has derived a reference dose of 0.1 mg/kg-day for chronic oral exposures to ethylbenzene based on a 182 day oral bioassay using female, albino rats. The reference dose includes an uncertainty factor of 1000 to account for the intraspecies and interspecies variability to the toxicity of this chemical in lieu of specific data, and for the extrapolation of a subchroinc effect level to its chronic equivalent (IRIS, 1988).

C. Pharmacokinetics

Ethyl benzene is absorbed after exposure by the inhalation, oral, or dermal route since toxic effects have been produced after exposure by these routes. By the dermal route, Dutkiewicz and Tyras (1968 as cited in U.S. EPA, 1980) found that ethyl benzene was absorbed at a rate of 0.11-0.21 mg/cm^2/hr in human subjects. The concentration of ethyl benzene used in the above experiment was 112-156 mg/l. The metabolism of ethyl benzene has been extensively investigated in experimental animals (U.S. EPA, 1980). The major metabolites are believed to be 1-phenylethanol, hippuric acid, and -phenaceturic acid (U.S. EPA, 1980). The major route by which ethyl benzene is eliminated is urinary excretion of its metabolites (U.S. EPA, 1980).

D. Environmental Effects

The available data for ethyl benzene indicate that acute toxicity to fresh water aquatic life occurs at concentrations as low as 32,800 ug/l. No definitive data are available concerning the chronic toxicity of ethyl benzene to sensitive fresh water life. Data concerning the toxicity of ethyl benzene to domestic animals or terrestrial wildlife was not available.

E. Guidelines and Standards

Water Quality (U.S. EPA, 1980) : For the protection of human health through ingestion of water and contaminated aquatic organisms, the ambient water quality criterion is 1.4 mg/l. For protection from contaminated aquatic organisms alone, the criteria is 3.28 mg/l.

OSHA: (skin) 435 mg/m^3 TWA.

Drinking Water: the MCLG for ethyl benzene is 0.68 mg/l (proposed, EPA,1985).

The EPA one-day and ten-day Drinking Water Health Advisories for a 10kg child are 32 mg/l and 3.2 mg/l, respectively. The lifetime Health Advisory for a 70kg person is 0.68 mg/l.

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XYLENE
(Dimethylbenzene, Xylol)

A. Physical and Chemical Properties

Property(a)	Value	Reference
Molecular Weight	106.16 g/mol	Verschueren, 1983
Water Solubility	175 mg/L at 20°C, insoluble, and 198 mg/l at 25°C for 1,2-, 1,3- and 1, 4-xylene	Verschueren, 1983
Vapor pressure	5,6, and 6.5 mm Hg for 1,2-, 1,3- and 1,4-xylene at 20°C	Verschueren, 1983
Koc	2.4×10^2 ml/g	
Kow	589, 1585, and 1412 for 1,2-, 1,3-, and 1,4-xylene	Lyman et. al., 1982
Hc	5.1×10^3 atm-m^3/mol	Lyman et. al., 1982

(a) Koc = organic carbon-water partition coefficient:
Kow = octanol-water partition coefficient:
Hc = Henry's Law constant:

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

When inhaled for short times at high concentrations, xylene causes CNS depression. It can also cause reddening of the face, disturbed vision, and salivation. There is some evidence suggesting that xylene sensitizes the myocardium to the endogenous neurohormone, epinephrine, and can precipitate heart failure and death.

Workers chronically exposed to xylene display symptoms similar to those seen in acutely exposed individuals (Sandmeyer, 1981). In addition, there have been reports that disturbances in the blood can occur from xylene exposure. These effects, however, may be due to benzene contamination. The ACGIH has recommended a TLV for xylene of 100 ppm (ACGIH, 1980).

There are no studies to indicate that xylene is carcinogenic or mutagenic. A two year bioassay did not reveal an increased incidence of cancer in rats and mice.

Marks et. al. (1982) reported that xylene caused adverse reproductive effects in mice. In this study, mice were administered xylene three times per day on days 6-15 of gestation by oral gavage at doses from 0.52 to 4.13 g/kg/day. An increase in cleft palate formation in the offspring was noted at doses of 2.06 g/kg/day and higher. At these doses, an increase in liver weight and a decrease in fetal weight were also observed. No adverse effects were seen at a dose of 1.03 g/kg/day. Mirkova et. al. (1983) reported that xylene caused embryotoxic effects in rates after inhalation exposure. In this study, rats were exposed to xylene at a concentration, of 10, 50, or 500 mg/m^3, 6 hr/day, 5 days/wk from days 1 through 21 of gestation. Significant embryotoxic effects were produced after exposure to the two higher concentrations but not to the lowest concentration.

The EPA has derived a reference dose of 2 mg/kg-day for chronic oral exposures to xylenes based on chronic bioassays using male and female rats and mice. The reference dose includes an uncertainty factor of 100 to account for the intraspecies and interspecies variability to the toxicity of this chemical.

C. Pharmacokinetics

Xylene is presumed to be absorbed after inhalation or oral exposure since toxicities have resulted after exposure by these routes. The rates at which xylene is absorbed have not been well characterized. The available data suggest that xylene toxicity is more severe after inhalation exposure than after oral exposure.

The major metabolite of xylene is methyl hippuric acid. This metabolite also represents the major urinary excretory product of xylene. Xylene can also be eliminated unchanged in the exhaled air.

D. Environmental Effects

Xylene is a volatile aromatic hydrocarbon. Because it readily volatilizes from surface water and surface soil environments, it tends not to be persistent. In aquatic environments some fraction of the chemical may become adsorbed onto organic particulate matter and settle to the bottom or remain in suspension. The chemical may be degraded by photodegradation processes in the atmosphere and shallow water or surface soil environments and by microorganisms in soil and water.

Because xylenes are volatile chemicals, static acute bioassays tend to underestimate their toxicity. Flow-through bioassays have been used to determine the acute toxicity of xylenes to trout and yielded an LC-50 value of 13,500 ug/l. . Acute toxicity of xylenes to marine organisms are similar though somewhat lower with LC-50 values in the range of 2,000 to 10,000 ug/l. No data were found on the chronic toxicity of xylene. Sublethal effects (avoidance) has been observed with fish larvae which responded to concentrations of xylene on the order of 100 ug/l.

Because of the volatility and low toxicity of xylenes, effects upon wild birds and mammals are expected to be minimal. No specific studies have been conducted to formally evaluate this.

E. Reported Levels

In Outdoor Air:	Concentration (ng/m3)	Reference (see Appendix A)
St. Louis	5,451	Singh et. al., 1983
Oakland	9,864	
Pittsburgh	10,120	
Chicago	10,406	
Riverside	14,411	
Denver	17,912	
Houston	22,269	
Staten Island	25,224	
Phoenix	25,872	
Los Angeles	28,295	
In Indoor Air:	(ng/m3)	
Elizabeth-Bayonne		
Winter	49,000	U.S. EPA, 1987
Summer	49,000	
Fall	68,000	
New Jersey		
Winter	38,800	
Summer	27,000	
Fall	71,000	
Los Angles		
February	41,000	
May	31,400	
Contra Costa		
June	14,700	

F. Guidelines and Standards

Air: The federal standard and the ACGIH, 1983/1984 TLV value is 100 ppm (435 mg/m^3) for all isomers. NIOSH recommends adherence to the present federal standard of 100 ppm as a time weighted average.

Water Quality: No criteria set by U.S. EPA.

Drinking Water: 440 ug/l has been proposed as a Maximum Contaminant Level Goal (MCLG);

The U.S.EPA one-day, ten-day, and longer-term Drinking Water Health Advisories for a 10kg person are 12,000 ug/l, 7,800 ug/l and 7,800 ug/l, respectively. The longer-term and lifetime Health Advisories for a 70kg person are 27,300 ug/l and 2,200 ug/l, respectively.

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ARSENIC

Arsenic exists as a nonmetal or metalloid that may be present in the environment as a constituent of organic and inorganic compounds. The complexing of arsenic with organic compounds increases its solubility potential. Naturally-occurring arsenic can exist in four oxidation states: -3, 0 (the metallic state), +3, and +5. Arsenate (As^{+5}) is the dominant species in aquatic systems. Biological activity may reduce arsenate to arsenite (As^{+3}) and finally to methylated arsenicals (As^{-3}) (Callahan et al, 1979).

The toxicity of arsenic depends upon its chemical form along with the route, dose, and duration of exposure. In general, arsenites (As^{+3}) are potentially more toxic than arsenates, soluble arsenic compounds are potentially more toxic than insoluble compounds, and inorganic arsenic compounds are potentially more toxic than organic derivatives (U.S. EPA, 1985b).

1.0 Human Health Effects

1.1 Summary of Toxicological Effects

1.1.1 Pharmacokinetics

Absorption from the gastrointestinal tract (orally) is dependent upon the solubility of the specific arsenic compound and the dose. Solutions of As^{+3} or As^{+5} soluble inorganic compounds are reported to be most completely absorbed by rats (Coulson et al, 1935). Ray-Bettley and O'Shea (1975) have reported that greater than 95% of the soluble inorganic arsenic that humans consume may be absorbed. Insoluble forms of arsenic tend to pass through the gastrointestinal tract (Mappes, 1977). In humans, the rate of arsenic excretion, which is dependent upon dose and oxidation state, is reported to be 50% to 90% within two to four days (Crecelius, 1977). Studies performed to determine the pharmacokinetics of arsenic have primarily used inorganic arsenic compounds. These compounds undergo methylation in mammalian species to organic arsenic compounds, monomethyl and dimethyl arsenic (Buchet, et al, 1981). Buchet et al (1981) reported that after oral administration of 500 ug of arsenite to humans, 25% was excreted in the urine in the inorganic form, 25% as monomethylarsenic acid and 50% in dimethyl form. Crecelius (1977)

reported urinary excretion after the ingestion of arsenic-rich wire to be approximately 80%: 63% dimethylarsenic acid, 18% monomethylarsenic acid and 9% of both inorganic AS^{+3} and As^{+5}. The primary route of excretion is reported to be via the urine, initially in the form of the ingested compounds followed by the metabolized methyl derivative forms (Crecelius, 1977).

Absorption from the respiratory tract is also dependent upon the specific arsenic compound, along with particle size (from aerosols or dusts). Particles less than 1 to 2 um may be absorbed through the respiratory epithelium; whereas, larger particles are most likely deposited in the upper respiratory tract and ultimately swallowed (U.S. EPA, 1984). For both forms of exposure, excretion is primarily through the urine.

1.1.2 Human Data

Acute Toxicity

Depending upon dose, arsenic is a potential irritant of the skin, mucous membranes, and the gastrointestinal tract. Acute toxicity from the ingestion of higher doses of arsenic may result in vomiting, diarrhea, convulsions, a severe drop in blood pressure, and cardiovascular effects. The lethal dose for humans is reported to be 70 to 180 mg or 1.0 to 2.6 mg/kg-bw for a 70 kg adult (Vallee et al, 1960). Acute toxicity from inhalation exposure to arsenic adsorbed to particulate matter (aerosols) may result in cojunctivitis and pharyngitis.

Subchronic Toxicity

Tay and Seah (1975) reported subchronic effects in patients in Singapore who had ingested arsenic-containing antiasthmatic herbal preparations at doses of 2.5 mg As/day as arsenic oxide (As^{+3}) or 10.3 mg As/day as arsenic sulfides over a period of less than 6 months to 15 years. The major effects reported included: hyperpigmentation (melanosis), multiple arsenical keratoses, sensorimotor polyneuropathy, persistent chronic headache, lethargy, gastroenteritis, and mild iron deficiency anemia. Inhaled arsenic compounds have been reported to be associated with skin lesions, cardiovascular and respiratory effects, and peripheral neuropathy at varying doses (Stokinger, 1981; IARC, 1980; ACGIH, 1980).

disorders, hyperpigmentation, and keratosis that may develop into basal or squamos cell carcinoma (U.S. EPA, 1985b).

Multiple skin cancers were reported in humans who had used Fowler's solution, a medicinal trivalent arsenical, treated with arsenic (Cuzick et al, 1982). Several epidemiological studies of workers occupationally exposed to arsenic in a copper smelting facility have reported a correlation between this exposure and mortality due to respiratory cancer (Brown and Chu, 1983; Higgins et al, 1982; Enterline and Marsh, 1982). Enterline and Marsh (1982) reported an increase in deaths due to respiratory cancer in smelter workers employed from 1940-1964. Brown and Chu (1983) reported an increasing risk or respiratory cancer at increasing age of initial exposure to arsenic at a copper smelting facility that was independent of the time when exposure ceased.

1.1.3 Animal Data

Acute Toxicity

Heywood and Sortwell (1979) administered 0, 2.8, or 5.6 mg/kg/day As^{+5} orally to 3 pairs of Rhesus monkeys. The monkeys that received the highest dose were sacrificed on day 13 for "humane" reasons and only one of the monkeys receiving 2.8 mg/kg/day As^{+5} was reported to have assumed an unnatural posture in one arm.

Subchronic Toxicity

Toxicity data from arsenic exposure to rats cannot be readily extrapolated to man as the rat is able to store this compound bound to hemoglobin in red blood cells (Lanz et al, 1950). This binding results in extremely slow excretion by rats compared to other species (Mealey et al, 1959). For this reason, dogs have been used to obtain experimental toxicity information. Subchronic oral toxicity of diets containing sodium arsenite or sodium arsenate fed to dogs report that arsenite is potentially more toxic than arsenate. The NOEL (no observed effect level) was reported to be 50 ppm in the diet (2.1 mg/kg-bw/day) for both substances (Byron et al., 1967).

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Chronic Toxicity

Schroeder and Balassa (1967) studies the chronic oral toxicity of arsenic on growth and survival in mice. Ingestion of water containing As^{+3} at 5 mg/L over two years is reported to have resulted in decreased survival and reduced median life span in male and female mice. No information regarding chronic inhalation exposure of experimental animals to arsenic could be located in the available literature.

Developmental and Reproductive Effects

Animal studies to test the teratogenic potential of arsenic have been performed. Most of these studies have involved the administration of As^{+3} to parent animals (Ferm and Carpenter, 1968; Holmberg and Ferm, 1969; Beaudion, 1974; Ferm, et al, 1977). Oral exposure has been reported to result in fetotoxic or teratogenic effects. Single oral doses of 29 mg/kg As^{+5} administered by gavage to pregnant mice on day 9, 10, or 11 of gestation were reported to result in 17% to 26% dead or resorbed fetuses, a 10% to 16% decrease in average fetal weight, and a 1% to 3% rate of malformed fetuses (Hood, et al, 1977). Matsumoto et al (1973) reported decreased fetal weight in oral doses of up to 40 mg-arsenate/kg-bw/day administrated to pregnant mice for three consecutive days. Diets containing up to 100 mg-arsenite/kg-diet, however, were reported to have had no effect on offspring (Kojima, 1974). No data regarding the teratogenicity of inhaled arsenic could be found in the literature.

Mutagenicity

Arsenic compounds test weak or negative in most bacterial assays for mutagenicity (Nishioka, 1975).

Carcinogenicity

The majority of tests in which experimental animals were exposed orally to a variety of arsenic compounds produced negative results regarding carcinogenicity (Hueper and Payne, 1962; Baroni et al, 1963; Byron et al, 1967). Hueper and Payne (1962) and Baroni et al (1963) reported no increase in the incidence of tumors in mice that were subjected to 0.0034% or 0.01% arsenic trioxide in drinking water. Negative results were also reported for a similar study in which 5 mg sodium arsenite per liter

drinking water was administered to mice (Kanisawa and Schroeder, 1967 and 1969). Another study in which dogs were fed diets containing 5-125 mg/kg-As for two years reported no tumorigenic activity (Byron, et al, 1967). A few studies have, however reported tumorigenic effects of arsenic treatment (Schrauzer et al, 1978, Knoth, 1966 and 1967). Mice administered Fowler's solution (1% arsenic trioxide) orally once/week for 5 months were reported to show an increase in adenocarcinomas of the skin, lung, peritoneum and lymph nodes (Knoth, 1966 and 1967). Schrauzer et al (1978) reported 2 mg As/L water increased the growth rate and incidence of multiple tumors in test animals already exhibiting tumorigenic effects. Mixed results were reported in arsenic inhalation studies (Ishinishi et al, 1976; Ivankovic et al, 1979). Shirachi et al, 1983; Kanisawa and Schroeder, 1967 and 1969. Ishinishi et al (1976) reported no significant increase of tumor incidence between test animals intratracheally administered 0.26 mg arsenic trioxide, 3.95% arsenic in copper ore, or 10.5% As in refinery blue condensate for 15 weeks over controls. Invankovic et al (1979), however, reported lung tumors in 9 out of 15 rats administered a single dose of 4% calcium arsenate intratracheally.

1.2 EPA Classification of Carcinogenicity

Based upon epidemiological data, the U.S. EPA has classified arsenic as Group A - Human Carcinogen.

1.3 Quantitative Indices of Toxicity

Arsenic has been classified as a human carcinogen, therefore, neither an AIS (acceptable intake subchronic) nor an AIC (acceptable intake chronic) has been established. A cancer potency factor of 15.0 (mg/kg/day)-1 , assuming a human intake of 2 L of water/day and 100% oral absorption, was calculated for oral exposure to arsenic based on a correlation between skin cancer and arsenic ingestion (Tseng et al, 1968). A factor of 50.1 (mg/kg/day)-1 has been set for inhalation exposure based on epidemiological data from copper smelter workers (Brown and Chu, 1983) and assuming 30% inhalation absorption and that a 70 kg human inhales 20 m3/day.

1.4 Regulatory Standards and Criteria

ACGIH (1980) has established a TWA of 0.2 mg/m3 for arsenic and soluble arsenic compounds. NIOSH (1975) recommended a TWA (15-minute ceiling) of 0.002 mg/m3. OSHA established a standard of 0.01 mg/m3 for airborne inorganic As (U.S. EPA, 1980b). U.S. EPA water quality criteria for human health are : 0 (2.2 ng/l) for the consumption of aquatic organisms and drinking water and 0 (25 ng/l) for drinking water only (U.S. EPA, 1986). Based on the nonthreshold concentration for potential carcinogens, zero concentration is recommended for maximum protection of human health, while the concentration in parentheses corresponds to a 10-6 excess lifetime cancer risk. The MCL (maximum contaminant level) in drinking water is 0.05 mg/l with an RMCL (recommended maximum contaminant level) of the same value.

2.0 Environmental Effects

2.1 Summary of Environmental Effects

The chemistry of arsenic in natural waters is complex. Four species of arsenic are common in natural waters, arsenate (V), arsenate (III), methanearsenic, and dimethylarsenic acid. In general, the pentavalent forms of arsenic are expected to be most prevalent in the aerobic neutral waters that are the habitat for most aquatic species (ADL,1982). The chemical and toxicological properties of the forms are different and the toxicities of the forms have not been shown to be additive (Callahan et al., 1979).

Most of the saltwater toxicity data is available for the trivalent form of arsenic. Fish species were least sensitive with a range of LC50's from 12,700 to 16,033 ug/l (U.S. EPA, 1983). Among the invertebrates, the copepod, Arcatia tonsa, was the most sensitive at 508 ug/l and the polychaete worm, N. arenaceodentata was the least sensitive at 10,120 ug/l.

The U.S. EPA (1987) summarizes data concerning the acute toxicity of twelve saltwater species to arsenic (III). These species included invertebrates filter feeders, deposit feeders, plankton shrimp, and higher predators, salmon. The acute toxic values ranged from 232 to 16,030 ug/l. The only available values for arsenic (V) are

for two invertebrates and are between 2,000 and 3,000 ug/l. The two forms of arsenic are equally toxic to various species of saltwater algae but the sensitivities range form 19 ug/l to more than 1,000 ug/l.

2.2 Regulatory Standards and Criteria

The U.S. EPA recommends that saltwater species and their uses should not be affected unacceptably if the 4 day average concentration of arsenic (III) does not exceed 36 ug/l more than once every 3 years on the average and if the 1 hour average concentration does not exceed 69 ug/l more than once every 3 years on the average.

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A. Physical and Chemical Properties

Lead is a heavy metal that exists in one of three oxidation states 0, +2, and +4. Metallic lead and common lead minerals are insoluble in water. However, organic lead compounds are soluble in water (Clement, 1985). Metallic lead is used as a major component of many alloys such as solder, type metal, and many bronzes. Lead compounds have a wide variety of uses especially as paint pigments, in storage batteries and ceramics (ACGIH, 1986).

Property	Value	Reference
Molecular Weight	207.2	Clement, 1985

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

Toxic effects resulting from chronic lead exposure are well documented and many have been associated with accompanying blood-lead (PbB) levels. Children have been found to develop symptoms at lower PbB levels than do adults. The most serious effects associated with lead intoxication are the neurotoxic effects. Lead encephalopathy can result from blood lead levels greater than 100 ug/100 ml and is characterized by irritability, loss of memory and ability to concentrate, delirium, hallucinations, cerebral edema, and coma (U.S. EPA, 1980). Less severe neurotoxic effects have been observed at lower blood lead levels. For example, lowered nerve conduction velocities, indicative of peripheral nerve dysfunction, have been noted in adults at blood levels of 30 to 40 ug/100 ml (U.S. EPA, 1985).

Hematologic effects appear to be among the most sensitive indicators of lead absorption. Lead interference with heme synthesis has been noted in humans and other mammalian species at levels below 10-15 ug/100 ml. The step most sensitive to lead in the heme synthetic pathway is that mediated by the enzyme 7-aminolevulinic acid dehydratase (7-ALAD), although the health significance of 7-ALAD inhibition at low blood-lead levels is unclear. Lead can also lead to the accumulation of porphyrin in erythrocytes with elevated levels of erythrocyte protoporphyrin (EP) associated with blood lead levels of 25-30 ug/100 ml in

adults and 15 ug/100 ml in children (U.S. EPA, 1985). Anemia is characteristic of more severe cases of lead, poisoning, resulting from erythrocyte destruction and reduced hemoglobin synthesis (U.S. EPA, 1977).

Renal toxicity has also been observed in victims of lead intoxication. Reversible proximal tubule damage has been observed primarily in cases of short-term exposure with reduced glomerular function associated with more chronic exposures (U.S. EPA, 1980). There is also evidence that lead affects other systems of the body. The gastrointestinal system is one of the earliest to show symptoms of lead intoxication with colic (acute abdominal pain) considered a consistent early symptom of lead poisoning. Colic is most often seen in cases of occupational lead exposure and has been reported in workers with blood lead levels as low as 30 ug/100 ml (U.S. EPA, 1983). Recently, environmental epidemiological studies have produced results suggestive of possible lead-induced reproductive effects. The authors of one study of women living within a 30- to 50-mile radius of a lead mining area of Missouri reported an increased incidence of premature fetal membrane rupture in term and preterm infants (17%), as compared to the rate reported for women living in an urban area of Missouri (0.44%). It was also reported that average maternal and fetal blood-lead levels in cases of membrane rupture were significantly higher than the average levels found for normal deliveries (U.S. EPA, 1980).

The authors of a similar study conducted in Sweden observed a significantly reduced average body weight in the offspring of employees and the inhabitants of two small industrial areas located near a smelter that emitted lead and other potentially hazardous substances, such as arsenic and sulfur dioxide. The authors also noted a higher frequency of spontaneous abortion in women working at the smelter during or before pregnancy and in the women living near the smelter (U.S. EPA, 1980).

A number of studies that involved cytogenetic investigations of the cells of lead-exposed workers have reported an increased incidence of chromosomal aberration. The results are conflicting, however, as a number of similar studies have reported negative results (IARC, 1982). Chromosomal aberrations have also been demonstrated in the lymphocytes from cynomolgus monkeys treated chronically with lead acetate. The aberrations were particularly severe in animals kept on low-calcium diets (U.S. EPA, 1983).

IARC (1982) concluded that there is inadequate evidence that lead and lead compounds cause cancer in humans. Epidemiology studies conducted on lead smelter workers, however, have found significant excesses of respiratory and digestive cancers but the workers were exposed to a number of toxic compounds thus preventing the establishment of a causal relationship. An insignificant increase in skin cancers has been noted in workers exposed to tetraethyl lead (IARC, 1982).

An RfD is based on the notion that a threshold exists below which adverse effects will not occur. The U.S. EPA has not established a risk reference dose (RfD) for lead because it appears that some of the observed effects occur at

such low doses as to be essentially without a threshold (IRIS, 1988). Nonetheless, under the Superfund program, the EPA did develop AIC values of 0.0014 mg/kg/day for lead via the oral route and 0.00043 mg/kg/day via the inhalation route (USEPA, 1986).

C. Pharmacokinetics

Lead can be absorbed through the gastrointestinal tract, the lungs, and the skin. It has been estimated that, on the average, 8 percent of normal adult dietary lead (including beverages) is absorbed. Recent studies indicate that the fraction of ingested lead that is absorbed is up to 8-fold higher when lead is ingested during periods of fasting than when it is when taken with meals. The gastrointestinal absorption of lead in young children is considerably greater than in adults, with about 50 percent of dietary lead absorbed. Numerous factors, including diet and the chemical nature of the lead, influence absorption of lead from the gastrointestinal tract. Absorption of lead from the respiratory tract depends on the solubility of the inhaled particles and particle size. Lead can also be absorbed through the intact skin, at least when applied at high concentrations. While quantitative data on dermal absorption of lead are not available, it has been found that organolead compounds (e.g., tetraethyl lead) are absorbed through the skin to a substantially greater degree than inorganic forms of lead (NAS, 1980; U.S. EPA, 1980).

In the body, about 94% of the adult body burden of lead is localized in the skeleton, about 4% is in the blood, and 2% is in soft tissues. In children, only about 72% of lead in the body is in the bone. Lead is known to cross the placenta, and thus concern exists over distribution during pregnancy and possible toxic effects on the fetus (NAS, 1980; U.S. EPA, 1980).

Upon entering the body, most lead compounds dissociate; in these cases, metabolism of lead is not an issue. An exception to this is the family of alkyl lead compounds (principally, tetramethyl lead and tetraethyl lead), which are dealkylated to form the more toxic tri- and dialkyl metabolites (U.S. EPA, 1980).

The two primary routes of excretion of lead from the body appear to be via the urine and the feces (U.S. EPA, 1980).

D. Environmental Effects

Lead occurs as free ion, organic and inorganic complexes, and associated with particles. With the exception of some organolead compounds, the free ion is the most toxic species in natural systems. The dominant form of lead in natural waters is the divalent cation, but its concentration is usually low because of precipitation by hydroxide and carbonate and sorption to organic matter (Callahan et. al., 1979). The concentration of soluble lead in natural waters is directly related to pH, oxidizing potential, the presence of competing ions (Ca++, Mg++, Fe++), and the existence of complexing agents such as the

organic acids humic and fulvic acid. Lead levels in sea water are typically 0.005 μg/l (ADL, 1982).

Marine organisms can bioaccumulate lead. Kopfler and Mayer (1973) measured bioconcentration factors (BCFs) in oysters (Crassostrea virginca) ranging from 200 to 1,760. Mussels had higher BCF values, 4,400 to 13,400 (Marletta et. al., 1979). Varanasi and Gnur (1978) and Reichert et. al. (1979) measured relatively low BCFs, 40 to 300 for Coho salmon. The U.S. EPA (1983) reports BCFs for marine bivalves which range from 17.5 for the quahog (Mercenaria mercenaria) to 2,570 for the blue mussel (Mytilus edulis).

As with most metals, lead does not biomagnify up the food chain. Burnet and Patterson (1980) noted a three order of magnitude decrease in values over a phytoplankton-macroinvertebrate-tuna food chain.

U.S. EPA (1987) indicates that acute toxicity data for 13 marine species ranges from 315 μg/l for the mummichog (Fundulus heteroclitus) to 27,000 for the soft shell clam (Mya arenaria). Weis and Weis (1976) found inhibition of fin regeneration in the mummichog at 1,000 μg/l. Delayed crab larval developoment occurs at concentrations as low as 50 μg/l (Benjits, 1975). Reish and Carr (1978) report suppressed reproduction of two polychaete species at 1,000 μg/l.

Acute tests were conducted at three different levels of water hardness with Daphnia magna (Chapman et. al. Manuscript), demonstrating that daphnids were three times more sensitive to lead in soft water. This agrees closely with the value reported for the same species in soft water by Biesinger and Christensen (l972). The data also indicate that lead is more toxic to the rainbow trout, fathead minnow, and bluegill in soft than in hard water. Hale (l977) conducted an acute exposure of rainbow trout to lead and obtained an LC-50 value of 8,000 μg/l. This value is almost seven times greater than the LC-50 value obtained for rainbow trout in soft water by Davies et. al. (l976). Hale did not report water hardness; however, alkalinity and pH were reported to be l05 mg/l and 7.3, respectively, which suggests that this water was probably harder than the soft water used by Davies et. al. (l976).

Rotifers tested for 96 hours in soft water by Buikema, et. al. (l974) were very resistant to lead; however, scuds were reported by Spehar et. al. (l978) and Call et. al. (l982) to be more sensitive to lead than any other freshwater animal species thus far tested. Also, in exposures lasting up to 28 days, the scud was far more sensitive to lead than a snail, cladoceran, chironomid, mayfly, stonefly, and caddisfly (Spehar et. al., l978; Biesinger and Christensen, l972; Anderson et. al., l980; and Nehring, l976). Pickering and Henderson (l966) found that lead chloride and lead acetate were about equally toxic to the fathead minnow in static tests in soft water. Wallen et. al. (l957) found that lead oxide is much less acutely toxic than lead nitrate to the mosquitofish in turbid water containing approximately 300,000 ug/l suspended clay particles.

Different species exhibit different sensitivities to lead, and many other factors may affect the results of tests of the toxicity of lead to aquatic species. Criteria can quantitatively take into account such a factor, however, only if enough data are available to show that the factor similarly affects the results of tests with a variety of species. Hardness is often thought of as having a major effect on the toxicity of lead, although the observed effect is probably due to one or more of a number of usually interrelated ions, such as hydroxide, carbonate, calcium, and magnesium.

Chapman et. al. (Manuscript) studied the chronic toxicity of lead to Daphnia magna at three different hardnesses. The daphnids were nearly ll times more sensitive to lead in soft water than in hard water. For the same species in a different soft water, a chronic value over four times higher was obtained by Biesinger and Christensen (l972) in a test in which the concentrations of lead were not measured. The chronic values of Chapman were regressed against hardness, but the slope of 2.35 was not significant.

A life-cycle test on lead in hard water was conducted by Borgmann et. al. (l978) with snails. These authors used biomass as their endpoint and reported that lead concentrations as low as l9 ug/l significantly decreased survival but not growth or reproduction. However, it was not at all clear how these investigators arrived at such a low effect concentration. This publication did, however, contain suitable information for determining a chronic value. Chronic limits were taken directly from the cumulative percent survival figure which showed no observed effect on survival at l2 ug/l and almost complete mortality at 54 ug/l. The chronic value for snails was therefore established at 25 ug/l, which is somewhat lower than the chronic value reported for daphnids in hard water.

Davies et. al. (l976) published results of an early life-stage test with rainbow trout in soft water. Even though this test was started with embryos and continued for l9 months after hatch, it could not be considered a life-cycle test because no reproduction occurred.

Spinal deformities have also been caused by lead in a life-cycle test with brook trout (Holcombe et. al., l976) and in early life-stage tests with rainbow trout, northern pike and walleye (Sauter et. al., l976). The chronic value obtained for rainbow trout by Sauter et. al. (l976) is somewhat higher than that chronic value derived from Davies et. al. (l976). Even though the hardnesses were about the same, differences could be due to differences in the length of exposure (2 months vs. l9 months).

In addition, Davies et. al. (l976) described the long-term effects on rainbow trout fry and fingerlings exposed to various concentrations of lead for l9 months in hard and soft water. Although these tests were neither life-cycle (no natural reproduction) nor early life-stage (no embryos exposed), they do provide valuable information concerning the relationship between water hardness and the chronic toxicity of lead to fish. During these l9-month exposures, 60 to l00 percent of the trout developed spinal deformities in hard water at measured lead concentrations of 850 ug/l and above. In the softwater exposure 44 to 97

percent of the trout developed spinal deformities in measured lead concentrations as low as 3l ug/l. These results strongly demonstrate that lead is more chronically toxic in soft water than in hard water.

Four fresh water invertebrate species have been exposed to lead (Borgmann et. al., l978; Spehar et. al., l978) and the bioconcentration factors ranged from 499 to l,700. Brook trout and bluegills were also exposed to lead (Holcombe et. al., l976) and calculated bioconcentration factors were 42 and 45 respectively.

Lead toxicity in water fowl through the ingestion of lead pellets is well documented. Lead toxicosis has been observed in plants from lead concentratoins ranging from 0.005 to 33,000 mg/l. Effects include growth stimulation (at low levels), growth inhibition,leaf yellowing, absicsion, inhibition of mitosis and chlorophyll synthesis, loss of turgor pressure and death.

E. Reported Levels

In Soils and Rocks (mg/kg) :

Typical values for rocks/sediments

Parameter	Typical Values for Uncontaminated Soils	Granite Rock	Shales	Deep-Sea Clays	Sand
Lead	0 - 500	15	20	80	7
Available Lead	0 - 200				
N.J. Background	1 - 180		N.J. DEP (1987)		
National Background	2 - 200		N.J. DEP (1987)		

F. Guidelines and Standards

Water Quality: U.S. EPA (1987) states that salt water organisms and their uses should not be affected unacceptably if the four-day average concentration of lead does not exceed 5.6 ug/l more than once over 3 years on the average and if the 1-hour average concentration does not exceed 140 ug/l more than once every 3 years on the average. The values for acute toxicity in fresh water is $e^{1.34\ln(\text{hardness})-2.014}$ ug/l, and the value for chronic toxicity is $e^{1.34\ln(\text{hardness})-5.24}$ ug/l.

Ambient Water Quality Criteria:
0.05 mg/l, based on ingesting aquatic organisms and drinking water (EPA, 1980)
0.05 mg/l adjusted for Drinking Water Only:(EPA, 1986)

Drinking Water:
0.05 mg/l Maximum Contaminant Level (MCL) (final)
0.02 mg/l Maximum Contaminant Level Goal (MCLG) (proposed, EPA, 1985)

Air:
EPA Clean Air Act NAAQS: 0.0015 mg/m^3 (90 day average)
NIOSH REL : <0.10 mg/m^3 10 hrTWA
ACGIH TLV: 0.15 mg/m^3 (inorganic dust and fumes, as Pb) 8 hr TWA
OSHA PEL: 0.05 mg/m^3 TWA

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ZINC

Summary

Ingestion of excessive amounts of zinc can cause fever, vomiting, and stomach cramps. Zinc oxide fumes can cause metal fume fever. Inhalation of mists or fumes may irritate the respiratory tract, and contact with zinc chloride may irritate the eyes and skin. High levels of zinc in the diet have been shown to retard growth and produce defective mineralization of bone.

Background Information

Zinc generally exists in nature as a salt with a valence of +2, although it is also found in four other stable valences.

CAS Number: 7440-66-6

Chemical Formula: Zn

IUPAC Name: Zinc

Chemical and Physical Properties

Atomic Weight: 65.38

Boiling Point: 907°C

Melting Point: 419.58°C

Specific Gravity: 7.133 at 25°C

Solubility in Organics: Soluble in acid and alkali

Vapor Pressure: 1 mm Hg at 487°C

Transport and Fate

Zinc can occur in both suspended and dissolved forms. Dissolved zinc may occur as the free (hydrated) zinc ion or as dissolved complexes and compounds with varying degrees of stability and toxicity. Suspended (undissolved) zinc may be dissolved following minor changes in water chemistry or may be sorbed to suspended matter. The predominant fate of zinc in aerobic aquatic systems is sorption of the divalent cation by hydrous iron and manganese oxides, clay minerals, and orgaic material. The efficiency of these materials in removing zinc from solution varies according to their compositions and concentrations; the pH and salinith of the water; the concentrations of complexing ligands; and the concentration of

zinc. Concentrations of zinc in suspended and bed sediments always exceed concentrations in ambient water. In reducing environments, precipitation of zinc sulfide limits the mobility of zinc. However, under aerobic conditions, precipitation of zinc compounds is probably important only where zinc is present in high concentrations. Zinc tends to be more readily sorbed at higher pH than lower pH and tends to be desorbed from sediments as salinity increases. Compounds of zinc with the commmon logands of surface waters are soluble in most neutral and acidic solutions, so that zinc is readily transported in most unpolluted, relatively organic-free waters.

The relative mobility of zinc in soil is determined by the same factors affecting its transport in aquatic systems. Atmospheric transport of zinc is also possible. However, except near sources such as smelters, zinc concentrations in air are relatively low and fairly constant.

Since it is an essential nutrient, zinc is strongly bio-accumulated even in the absence of abnormally high ambient concentrations. Zinc does not appear to be biomagnified. Although zinc is actively bioaccumulated in aquatic systems, the biota appear to represent a relatively minor sink compared to the sediments. Zinc is one of the most important metals in biological systems. Since it is actively bioaccumulated, the environmental concentrations of zinc probably ehibit seasonal fluctuations.

Health Effects

Testicular tumors have been produced in rats and chickens when zinc salts are injected intratesticularly, but not when other routes of administration are used. Zinc may be indirectly important with regard to cancer since its presence seems to be necessary for the growth of tumors. Laboratory studies suggest that although zinc-deficient animals may be more susceptible to chemical induction of cancer, tumor growth is slower in these animals. There is no evidence that zinc deficiency has any etiological role in human cancer. There are no data available to suggest that zinc is mutagenic or teratogenic in animals or humans.

Zinc is an essential trace element that is involved in enzyme functions, protein synthesis, and carbohydrate metabolism. Ingestion of excessive amounts of zinc may cause fever, vomiting, stomach cramps, and diarrhea. Fumes of freshly formed zinc oxide can penetrate deep into the alveoli and cause metal fume fever. Zinc oxide dust does not produce this disorder. Contact with zinc chloride can cause skin and eye irritation. Inhalation of mists or fumes may irritate the respiratory and gastrointestinal tracts. Zinc in excess of 0.25% in the diet of rats causes growth retardation, hypochromic anemia, and defective mineralization of bone. No zinc toxicity is observed at dietary levels below 0.25%.

Studies with animals and humans indicate that metabolic changes may occur due to the interaction of zinc and other metals in the diet. Exposure to cadmium can cause changes in the distribution of zinc, with increases in the liver and kidneys, organs where cadmium also accumulates. Excessive intake of zinc may cause copper deficiencies and result in anemia. Interaction of zinc with iron or lead may also lead to changes that are not produced whten the metals are ingested individually.

Toxicity to Wildlife and Domestic Animals

Zinc produces acute toxicity in freshwater organisms over a range of concentrations from 90 to 58,100 ug/liter and appears to be less toxic in harder water. Acute toxicity is similar for freshwater fish and invertebrates. Chronic toxicity values range from 47 to 852 ug/liter and appear to be relatively unaffected by hardness. A final acute-chronic ration for freshwater species of 3.0 has been reported. Although most freshwater plants appear to be insensitive to zinc, one species, the alga Selenastrum capricornutum, exhibited toxic effects at concentrations from 30 to 700 ug/liter. Reported acute toxicity values range from 2,730 to 83,000 ug/liter for saltwater fish and from 166 to 55,000 ug/liter for invertebrate saltwater species. Zinc produces chronic toxicity in the ysid shrimp at 166 ug/liter. The final acute-chronic ratio for saltwater species is 3.0. Toxic effects are observed in saltwater plant species at zinc concentrations of 50 to 25,000 ug/liter. Bio-concentration factors of edible portions of aquatic organisms range from 43 for the soft-shell clam to 16,700 for the oyster.

Zinc poisoning has occurred in cattle. In one outbreak, poisoning was caused by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of 140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited severe enteritis, and some died or had to be slaughtered. Postmortem findings showed severe pulmonary emphysema with changes in the myocardium, kidneys, and liver. Zinc concentrations in the liver were extremely high. Based on relatively limited data, some researchers have speculated that expousre to excessive amounts of zinc may constitute a hazard to horses. Laboratory studies and findings in foals living near lead-zinc smelters suggest that excessive expoosure to zinc may produce bone changes, joint afflictions, and lameness. In pigs given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints. High concentrations of zinc were found in the liver.

Regulations and Standards

Ambient Water Quality Criteria (USEPA):

Aquatic Life

Freshwater

Acute toxicity: $e^{(0.83[\ln(\text{hardness})] + 1.95)}$ ug/liter
Chronic toxicity: 47 ug/liter

Saltwater

Acute toxicity: 170- ug/liter
Chronic toxicity: 58 ug/liter

Human Health

Organoleptic criterion: 5 mg/liter

Secondary Drinking Water Standard: 5 mg/liter

NIOSH Recommended Standard: 5 mg/m^3 (zinc oxide)

OSHA Standard: 5 mg/m^3 TWA (zinc oxide)

ACGIH Threshold Limit Values:

Zinc Chloride fume: 1 mg/m^3 TWA

2 mg/m^3 STEL

Zinc oxide fume: 5 mg/m^3 TWA

10 mg/m^3 STEL

Zinc oxide dust: 10 mg/m^3 TWA (nuisance particulate)

Zinc stearate: 10 mg/m^3 TWA (nuisance particulate)

20 mg/m^3 STEL

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TETRACHLOROETHYLENE

(Perchloroethylene, Tetrachlorethene, PCE, Perc)

A. Physical and Chemical Properties

Tetrachloroethylene is a halogenated aliphatic hydrocarbon. It is a non-flammable colorless liquid that is soluble in water and common organics. It is widely used as a solvent in degreasing operations and in dry cleaning.

Property (a)	Value	Reference
Molecular Weight:	166	US EPA, 1986
Vapor pressure	1.78×10^{1}mm Hg	US EPA, 1986
Water solubility	1.5×10^{2} mg/l	US EPA, 1986
Henry's law	2.59×10^{-2}atm-m^3/mol	US EPA, 1986
Koc	364ml/g	US EPA, 1986
log Kow	2.6	US EPA, 1986

(a) Koc=organic carbon-water partition coefficient;
Kow=octanol-water partition coefficient;
Hc=Henry's Law constant.

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S.EPA.

B. Health Effects

Pharmacokinetics

Tetrachloroethylene is easily absorbed following dermal, gastrointestinal, and inhalation exposures (EPA, 1985). In humans, inhalation is the most common route of exposure. After absorption, tetrachloroethylene is distributed throughout the body. The highest concentration can be found in adipose tissue, brain, and the liver. Tetrachloroethylene metabolism is dose-dependent and saturable. Low percentages of the absorbed dose are excreted as metabolites such as trichloroacetic acid or perchloroethylene epoxide. Excretion takes place through the pulmonary route and consists mainly of the unchanged compound (EPA, 1985).

Systemic Effects - Humans

Systemic health effects have been observed in certain occupational settings where tetrachloroethylene was used as a solvent. OSHA standards (see Section E of this profile) have been established to protect against these exposure levels. Based on occupational exposures it has been observed that acute (short-term) inhalation exposure to elevated levels of tetrachloroethylene in air can cause symptoms such as dizziness, confusion, nausea, headache, and irritation of the eyes and mucous tissue. As concentrations continue to increase in air, these symptoms increase and eventually produce faintness and unconsciousness. Chronic (long-term) exposure has been observed to enhance the symptoms. Chronic exposure to elevated levels of tetrachloroethylene in air can also affect short-term memory and produce disorientation, irritability, ataxia, and sleep disturbance. Other reported effects include liver cirrhosis, hepatitis, and nephritis (EPA, 1985). All the observed effects associated with tetrachloroethylene in air are associated with levels of the chemical that are well above OSHA standards and many orders of magnitude greater than levels typically found in water that has been contaminated with the chemical.

Systemic Effects - Animals

Effects observed in experimental animals are similar to those reported from human acute and chronic exposures at both low and high concentrations of tetrachloroethylene. The acute oral toxicity has been determined for rats, mice, cats, rabbits, and dogs. No subchronic oral exposure studies exist, and only one chronic study (EPA, 1986).

Carcinogenicity

Tetrachloroethylene is classified as a Group B2/C carcinogen by the EPA. This reflects the uncertainty as to whether the compound is a probable human carcinogen (i.e., a B2 or higher classification) or only a possible carcinogen (C). At present, it is proposed to be regulated as a probable carcinogen even though the evidence to support this is not strong. The classification of this substance is currently under review. There is no adequate evidence from studies on humans demonstrating the carcinogenicity of tetrachloroethylene. Whether the evidence of carcinogenicity in experimental animals from tetrachloroethylene is "limited" or "adequate" is under review within EPA.

One oral study, conducted by the National Cancer Institute, showed a statistically significant increase in the incidence of hepatocellular carcinomas when tetrachloroethylene was

administered by gavage to male and female mice. The data on rats in this study was inconclusive due to early mortality (EPA, 1985; ATSDR, 1988).

In an inhalation bioassay conducted by the National Toxicology Program, tetrachloroethylene induced an increased incidence of mononuclear cell leukemia in female and male rats, although the results in males were only marginally statistically significant. Tetrachloroethylene treated male rats also demonstrated a dose related, but not significant, increase of rare renal tubular cell adenomas and/or carcinomas. As in the oral bioassay, statistically significant increases in liver tumors were observed in male and female mice exposed by inhalation to tetrachloroethylene (EPA, 1986; ATSDR, 1988).

Mutagenicity

Tetrachloroethylene has been studied using a variety of in vivo and in vitro test systems. For the most part, the results reported have been negative. A few positive results have been observed, but problems with purity of the compound and study design flaws make the results difficult to interpret (EPA, 1986).

Reproductive Effects

A developmental study has been conducted in both rats and mice exposed to tetrachloroethylene via inhalation. Tetrachloroethylene has been found to be fetotoxic, but not teratogenic at concentrations that are also maternally toxic. In mice exposed to tetrachloroethylene via inhalation, there is some indication that sperm abnormalities may occur (ATSDR, 1988).

C. Health Criteria

The EPA has developed an oral risk reference dose of 0.01 mg/kg/day. This value is based on the no observed adverse effect level of 14 mg/kg/day in a six week study in mice. At higher doses, liver toxicity was observed. The risk reference dose includes an uncertainty factor of 1000 to account for interspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent (IRIS, 1988).

The EPA Carcinogen Assessment Group has calculated carcinogenic potency factors for tetrachloroethylene by oral and inhalation exposures as: 5.1×10^{-2} and 1.7×10^{-3} $(\text{mg/kg/day})^{-1}$, respectively (EPA, 1986).

D. Environmental Effects

Available data indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 5,280 and 840 ug/l, respectively, and could occur at lower concentrations among species that are more sensitive than those tested.

Tetrachloroethylene is not expected to be persistent in the tissues of aquatic animals or to be bioaccumulated to any extent. The chemical is not expected to be tranferred via food webs.

E. Guidelines and Standards

Water

Human Health

USEPA Health Advisories (1987a) based on non-carcinogenic effects
Drinking Water Health Advisory,long-term (child): 1,400 ug/l
Drinking Water Health Advisory,long-term (adult): 5,000 ug/l

Proposed Maximum Contaminant Level: 5 ug/l
(based on the presumption that tetrachloroethylene is a probable carcinogen; as noted above, there is considerable uncetainty concerning the possible carcinogenicity of this compound);

Aquatic Biota

No criteria have been developed because of the limited data base. However "Lowest Observed Effects Levels" (LOELs) have been observed at 5,200 ug/l for acute exposure and 840 ug/l for chronic exposure. These values, with "uncertainty" or "safety" factors applied, provide a basis for judging the environmental significance of tetrachloroethylene in surface water.

Air

ACGIH TLV-TWA	340 mg/m^3
ACGIH STEL	1340 mg/m^3
OSHA PEL (1988)	670 mg/m^3 8 hr TWA
OSHA PEL (proposed, 1989)	170 mg/m^3 8 hr TWA

F. Occurrence in Environment

Tetrachloroethylene has been used widely in the United States as a solvent for domestic and commercial applications. As a result, the chemical can typically be found at low levels (ppb) in ambient air, indoor air, and many public water supplies.

Ambient Air Levels (ug/m^3) (Singh et al, 1983)

City	Average Concentration
Pittsbugh	2.7
Chicago	3.4
Staten Island	2.0
Riverside, CA	3.2
Denver	2.7
St. Louis	2.2
Houston	2.7
Oakland, CA	2.1
Phoenix, AZ	6.7
Los Angeles	10.0

Indoor Air Residential Levels (ug/m^3) (USEPA, 1987b)

Location	Median Concentration
New Jersey Homes (1981)	6.4
New Jersey Homes (1982)	5.5
New Jersey Homes (1983)	6.6
California Homes (1984)	8.3
California Homes (1984)	1.9
California Homes (1984)	1.8

Drinking Water Supplies

Tetrachloroethylene has been found in a number of municipal water supplies throughout the United States. It has been found in finished water of the New Orleans area at up to 5 ug/l (USEPA, 1974) and in eight of ten water utilities surveyed by the USEPA (1975) at levels of 0.07-0.46 ug/l.

Surveys of drinking water supplies in the United States have found that 3% of all public systems derived from well water contain tetrachloroethylene at levels of 0.5 ug/l or higher (USEPA 1987a). A small number of systems (0.7%) have levels higher than 5 ug/l.

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CHRYSENE

A. Physical and Chemical Properties

Property	Value	Reference
Molecular weight	228.3 g/mol	Verschueren, 1983
Water solubility	6.0×10^{-3} ppm at 25°	" "
Vapor pressure	6.9×10^{-9} torr at 25°C	Mabey et al., 1982
Koc	2.0×10^{5}	" " "
Kow	4.1×10^{5}	" " "
Hc	1.05×10^{-6} atm-m^3/mol	" " "

The values for the physical and chemical properties of various compounds are reported in the literature by various sources. Values differ typically because the experiments used to determine them were performed under diverse conditions (e.g. temperature). For more information about the properties of various compounds, the investigator should consult different data bases, such as the Integrated Risk Information System (IRIS) that is available from EPA.

B. Toxic Effects to Humans

There are no data on the toxicity of chrysene to man (IARC, 1983). The relative tumorigenic potency of chrysene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays tested using similar protocols (U.S. EPA, 1984). The ranking was as follows: benzo(a)pyrene > dibenzo(a,h)anthracene > benzo(b)fluoranthene > benzo(a)anthracene > indeno(1,2,3-c,d)pyrene > chrysene.

There is limited evidence that chrysene is mutagenic in short-term assays (IARC, 1983). There is no experimental data on the teratogenicity of chrysene in mammals.

IARC (1983) concluded that there is limited evidence to indicate that chrysene is carcinogenic in experimental animals. EPA's CAG has not estimated a CPF for chrysene (U.S. EPA, 1985). Based on the data of Wynder and Hoffman (1959), Clement Associates (1987)

estimated that chrysene had a relative potency, to B(a)P, of approximately 0.0044. This number can be used in the relative potency method.

C. Pharmacokinetics

There are no pharmacokinetic data for chrysene in man (U.S. EPA, 1980).In general, many PAHs can produce toxicity after inhalation, oral or dermal exposure. Thus, it is believed that PAHs are absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed-function-oxidase system, and elimination most likely occurs through the hepatobiliary tract.

Several monohydroxyl and dihydrodiol derivatives of chrysene have been reported (Sims, 1970; Nordquist et al., 1981; and Jacob et al., 1982, as cited in IARC, 1983). Epoxides of the 1,2-dihydrodiol and 3,4-dihydrodiol have also been reported (Chou et al., 1981; Nordquist et al., 1981; and Vyas et al., 1982, as cited in IARC, 1983). The 1,2-dihydrodiol and 1,2-diol-3,4-epoxide have been shown to be mutagenic in bacterial and mammalian cells (Wood et al., 1977; 1979, as cited in IARC, 1983) and inducers of pulmonary adenomas in newborn mice (Buening et al., 1979; and Chang et al., 1983, as cited in IARC, 1983). In addition, the 1,2-dihydrodiol has been shown to be a tumor-initiating agent on mouse skin (Levin et al., 1978; Slags et al., 1980; and Chang et al., 1983, as cited in IARC, 1983). The 1,2-diol-3,4-epoxide is believed to be the metabolite of chrysene that forms adducts with DNA (Hodgson et al., 1982; and Vigny et al., 1982, as cited in IARC, 1983).

D. Environmental Effects

The B(a)P environmental profile discusses the general environmental effects of PAHs.

As with many other individual PAHs, no specific information was obtained on the aquatic toxicity of this chemical. However, it is an animal-positive carcinogen. Chrysene produced skin tumors in mice following repeated paintings. High doses (2-20 mg) given by subcutaneous injection to mice produced a low incidence of tumors. Thus, chrysene, like other PAHs, may have carcinogenic or mutagenic effects on aquatic and terrestrial organisms. Adequate data for the charcterization of toxicity in domestic animals or wildlife is not available.

E. Reported Levels

In soil:	Concentration (mg/kg)	Reference (see Appendix A)
(unspecified)	0.04-0.24	IARC, 1973
urban	20	White and Vanderslice, 1980

In sediments:	(mg/kg)	
Heron I./Queensland	.0017	Smith et al., 1985
Burdekin R./G B reef	.0036	" " "
Gladstone H./Queensland	.0110	" " "
Ross R./G B reef	.0150	" " "
Buzzards Bay, New Bedford, MA	.0400	Giger and Blumer, 1974
Penobscot Bay, ME, outer region	.0598	Johnson et al., 1985
USA/general	.1400	Smith et al., 1985
New York Bight	.1409	Reid et al., 1982
Penobscot Bay, ME, inner region	.1969	Johnson et al., 1985
Buzzards Bay, New Bedford, MA	.2400	Giger and Blumer, 1974
Townsville H./Queensland	.4577	Smith et al., 1985
Boston Harbor	.7840	MA DEQE, 1985
Australia/general	1.5000	Smith et al., 1985
Long Island Sound	1.6685	Reid et al., 1982
Savern Estuary	2.9	John et al., 1979
Newton Creek, NY	3	Anderson, 1982
Commencement Bay, WA	4	Malins et al., 1982
Elliot Bay Pier 54, WA	6	" " "
Mystic River Boston, MA	9.5000	MA DEQE, 1985
Elizabeth River, VA	19	Bieri et al. (In press)

In air:	(ng/m^3)	
(unspecified)	0.2-10	U.S. EPA, 1982
urban areas	0.6-4.8	White and Vanderslice, 1980
U.S. cities	0.2-6.4	Gordon and Bryan, 1973
urban (summer)	2.5-3.6	IARC, 1973
urban (winter)	2.0-361	" "

F. Guidelines and Standards

Water quality: U.S. EPA (1986) has recommended criteria related to specific incremental lifetime risk levels of 10-5, 10-6 and 10-7. The corresponding water concentrations assuming ingestion of water and aquatic organisms, are 28, 2.8 and 0.28 ng/l, respectively. If these estimates are made for the consumption of aquatic organisms only, the levels are 311, 31.1 and 3.1 ng/l, respectively. The values acknowledge the conservative assumption that all carcinogenic PAHs are equal in potency to B(a)P. The carcinogenic potency of B(a)P and other PAHs is currently being re-evaluated by several industry groups and EPA. With regard to the protection of aquatic life, no numerical criteria have been developed.

OSHA limit: An 8-hour TWA concentration limit of 0.2 mg/m^3 has been set for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, B(a)P, phenanthrene, acridine, chrysene and pyrene).

NIOSH recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample determined as a 10-hour TWA.

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1,1,1-TRICHLOROETHANE
(methylchloroform, chlorothene, chloroethene, aerothene TT)

A. Physical and Chemical Properties

1,1,1-Trichloroethane is a volatile chlorinated hydrocarbon used extensively as an industrial solvent and in comsumer products, such as spot removers. It has also been used as an anesthetic (EPA, 1984).

Property(a)	Value	Reference
Molecular Weight	133 g/mol	US EPA, 1986
Water Solubility	1.5×10^3 mg/l	US EPA, 1986
Vapor Pressure	123 mm Hg	US EPA, 1986
Koc	152 ml/g	US EPA, 1986
log Kow	2.5	US EPA, 1986
Hc	1.44×10^{-2} atm-m^3/mol	US EPA, 1986

(a) Koc = organic carbon-water partition coefficient;
Kow = octanol-water partition coefficient;
Hc = Henry's Law constant

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

1,1,1-trichloroethane (TCA) has been tested in controlled exposure studies on humans. Exposures to 500 ppm or less in the air for 7.5 hours/day, 5 days/week for 3 weeks resulted in no serious deleterious effects on performance, and no abnormalities in various clinical tests. However, there was an obvious odor and slight changes in perception. (The odor threshold for TCA is 100 ppm). Fifteen minute exposures to concentrations above 1000 ppm result in mild eye irritation,.slightly longer or higher exposures result in lightheadedness and throat irritation (USEPA, 1984).

The EPA has determined a reference dose (RfD) for chronic oral exposure to 1,1,1-trichloroethane of 0.09 mg/kg-day. The RfD is based on a 6 month inhalation study of guinea pigs perfomed by Torkelson et al. (1958). Guinea pigs were the most sensitive species tested that also included rats, rabbits and monkeys. At 500 ppm, groups of 8 male and 8 female guinea pigs showed no evidence of adverse effects compared with unexposed and air exposed

controls. The EPA considered 500 ppm to be a no observed adverse effect level (NOAEL). The RfD was derived by converted the NOAEL to a dose and applying an uncertainty factor of 1000 (IRIS, 1988).

There is no human epidemiological evidence that TCA is carcinogenic. The animal bioassays conducted have yielded negative results, but are considered inconclusive by the EPA because of methodological flaws (EPA, 1984).

Commercially available samples of TCA are genotoxic to mouse hepatocytes and are wealkly mutagenic in an Salmonella (Ames) assay (EPA, 1984).

C. Pharmacokinetics

Inhalation, ingestion, and dermal exposures can result in the absorption of 1,1,1-trichloroethane. Inhalation is the most common exposure route for TCA and absorption is rapid. The percent of inhaled TCA that is retained by the body depends on various conditions, but appears to be 25-30% once equilibrium conditions are attained. TCA is also rapidly absorbed from the gastrointestinal tract, but only slowly and poorly absorbed through intact skin (EPA, 1984).

1,1,1-Trichloroethane distrubutes throughout the body, readily crossing the blood-brain barrier, and possibly the placental barrier as well. EPA considers that ATCA may also distribute into the colostrum or milk of nursing mothers, although no specific data are available (EPA, 1984).

1,1,1-Trichloroethane is metabolized to only a limited extent (3-6%) by mammals, including humans. The metabolites, including trichloroethanol and trichloroacetic acid, are excreted in the urine. Unmetabolized TCA is primarily excreted in expired air, for all routes of exposure (EPA, 1984).

D. Environmental Effects

1,1,1-Trichloroethane has been tested in a limited number of acute aquatic assays. The 48 hour LC50 value for Daphnia magna was greater than the highest exposure concentration of 530,000 µg/l. Acute toxicity tests with fathead minnows under static and flowthrough conditions yielded LC_{50}'s of 105,000 µg/l and 52,800 µg/l, respectively. The 96 hour (static) LC50 with bluegill was 69,700 µg/l (EPA, 1980). No chronic tests for 1,1,1-trichloroethane were found, although for 1,1,2-trichloroethane chronic toxicity was observed as low as 9400 µg/l (EPA, 1980).

TCA was tested 96 hour EC_{50} test with the green alga, Selenastrum capricornutum. However, the highest concentration used, 669,000 µg/l, was not high enough to yield the EC_{50} (EPA, 1980).

The steady state bioconcentration factor (BCF) for TCA in bluegill was measured to be 9. The elimination halflife is less than two days (EPA, 1980).

E. Guidelines and Standards

Ambient Water Quality Criteria:
1.84×10^4 µg/l based on ingesting aquatic organisms and drinking water (EPA, 1986)
1.9×10^4 µg/l adjusted for drinking water only (EPA, 1986)

Drinking water:		
MCLG	200 µg/l	final, EPA, 1985
MCL	200 µg/l	final, EPA, 1987
Health Advisories:		
1 day (10 kg child)	1.4×10^5 µg/l	EPA, 1986
10 day (10 kg child)	3.5×10^4 µg/l	EPA, 1986
lifetime (70 kg adult)	1×10^3 µg/l	EPA, 1986

Air:		
NIOSH REL	350 ppm TWA	NIOSH, 1985
OSHA PEL	350 ppm TWA	
ACGIH TLV	350 ppm TWA	

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TETRACHLOROETHYLENE
(Perchloroethylene, Tetrachlorethene, PCE, Perc)

A. Physical and Chemical Properties

Tetrachloroethylene is a halogenated aliphatic hydrocarbon. It is a non-flammable colorless liquid that is soluble in water and common organics. It is widely used as a solvent in degreasing operations and in dry cleaning.

Property (a)	Value	Reference
Molecular Weight:	166	US EPA, 1986
Vapor pressure	1.78×10^{1} mm Hg	US EPA, 1986
Water solubility	1.5×10^{2} mg/l	US EPA, 1986
Henry's law	2.59×10^{-2} atm-m^3/mol	US EPA, 1986
Koc	364ml/g	US EPA, 1986
log Kow	2.6	US EPA, 1986

(a) Koc=organic carbon-water partition coefficient;
Kow=octanol-water partition coefficient;
Hc=Henry's Law constant.

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S.EPA.

B. Health Effects

Pharmacokinetics

Tetrachloroethylene is easily absorbed following dermal, gastrointestinal, and inhalation exposures (EPA, 1985). In humans, inhalation is the most common route of exposure. After absorption, tetrachloroethylene is distributed throughout the body. The highest concentration can be found in adipose tissue, brain, and the liver. Tetrachloroethylene metabolism is dose-dependent and saturable. Low percentages of the absorbed dose are excreted as metabolites such as trichloroacetic acid or perchloroethylene epoxide. Excretion takes place through the pulmonary route and consists mainly of the unchanged compound (EPA, 1985).

Systemic Effects - Humans

Systemic health effects have been observed in certain occupational settings where tetrachloroethylene was used as a solvent. OSHA standards (see Section E of this profile) have been established to protect against these exposure levels. Based on occupational exposures it has been observed that acute (short-term) inhalation exposure to elevated levels of tetrachloroethylene in air can cause symptoms such as dizziness, confusion, nausea, headache, and irritation of the eyes and mucous tissue. As concentrations continue to increase in air, these symptoms increase and eventually produce faintness and unconsciousness. Chronic (long-term) exposure has been observed to enhance the symptoms. Chronic exposure to elevated levels of tetrachloroethylene in air can also affect short-term memory and produce disorientation, irritability, ataxia, and sleep disturbance. Other reported effects include liver cirrhosis, hepatitis, and nephritis (EPA, 1985). All the observed effects associated with tetrachloroethylene in air are associated with levels of the chemical that are well above OSHA standards and many orders of magnitude greater than levels typically found in water that has been contaminated with the chemical.

Systemic Effects - Animals

Effects observed in experimental animals are similar to those reported from human acute and chronic exposures at both low and high concentrations of tetrachloroethylene. The acute oral toxicity has been determined for rats, mice, cats, rabbits, and dogs. No subchronic oral exposure studies exist, and only one chronic study (EPA, 1986).

Carcinogenicity

Tetrachloroethylene is classified as a Group B2/C carcinogen by the EPA. This reflects the uncertainty as to whether the compound is a probable human carcinogen (i.e., a B2 or higher classification) or only a possible carcinogen (C). At present, it is proposed to be regulated as a probable carcinogen even though the evidence to support this is not strong. The classification of this substance is currently under review. There is no adequate evidence from studies on humans demonstrating the carcinogenicity of tetrachloroethylene. Whether the evidence of carcinogenicity in experimental animals from tetrachloroethylene is "limited" or "adequate" is under review within EPA.

One oral study, conducted by the National Cancer Institute, showed a statistically significant increase in the incidence of hepatocellular carcinomas when tetrachloroethylene was

administered by gavage to male and female mice. The data on rats in this study was inconclusive due to early mortality (EPA, 1985; ATSDR, 1988).

In an inhalation bioassay conducted by the National Toxicology Program, tetrachloroethylene induced an increased incidence of mononuclear cell leukemia in female and male rats, although the results in males were only marginally statistically significant. Tetrachloroethylene treated male rats also demonstrated a dose related, but not significant, increase of rare renal tubular cell adenomas and/or carcinomas. As in the oral bioassay, statistically significant increases in liver tumors were observed in male and female mice exposed by inhalation to tetrachloroethylene (EPA, 1986; ATSDR, 1988).

Mutagenicity

Tetrachloroethylene has been studied using a variety of in vivo and in vitro test systems. For the most part, the results reported have been negative. A few positive results have been observed, but problems with purity of the compound and study design flaws make the results difficult to interpret (EPA, 1986).

Reproductive Effects

A developmental study has been conducted in both rats and mice exposed to tetrachloroethylene via inhalation. Tetrachloroethylene has been found to be fetotoxic, but not teratogenic at concentrations that are also maternally toxic. In mice exposed to tetrachloroethylene via inhalation, there is some indication that sperm abnormalities may occur (ATSDR, 1988).

C. Health Criteria

The EPA has developed an oral risk reference dose of 0.01 mg/kg/day. This value is based on the no observed adverse effect level of 14 mg/kg/day in a six week study in mice. At higher doses, liver toxicity was observed. The risk reference dose includes an uncertainty factor of 1000 to account for interspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent (IRIS, 1988).

The EPA Carcinogen Assessment Group has calculated carcinogenic potency factors for tetrachloroethylene by oral and inhalation exposures as: 5.1×10^{-2} and 1.7×10^{-3} $(\mathrm{mg/kg/day})^{-1}$, respectively (EPA, 1986).

D. Environmental Effects

Available data indicate that acute and chronic toxicity to freshwater aquatic life occurs at concentrations as low as 5,280 and 840 ug/l, respectively, and could occur at lower concentrations among species that are more sensitive than those tested.

Tetrachloroethylene is not expected to be persistent in the tissues of aquatic animals or to be bioaccumulated to any extent. The chemical is not expected to be tranferred via food webs.

E. Guidelines and Standards

Water

Human Health

USEPA Health Advisories (1987a) based on non-carcinogenic effects
Drinking Water Health Advisory, long-term (child): 1,400 ug/l
Drinking Water Health Advisory, long-term (adult): 5,000 ug/l

Proposed Maximum Contaminant Level: 5 ug/l
(based on the presumption that tetrachloroethylene is a probable carcinogen; as noted above, there is considerable uncetainty concerning the possible carcinogenicity of this compound);

Aquatic Biota

No criteria have been developed because of the limited data base. However "Lowest Observed Effects Levels" (LOELs) have been observed at 5,200 ug/l for acute exposure and 840 ug/l for chronic exposure. These values, with "uncertainty" or "safety" factors applied, provide a basis for judging the environmental significance of tetrachloroethylene in surface water.

Air

ACGIH TLV-TWA	340 mg/m^3
ACGIH STEL	1340 mg/m^3
OSHA PEL (1988)	670 mg/m^3 8 hr TWA
OSHA PEL (proposed, 1989)	170 mg/m^3 8 hr TWA

F. Occurrence in Environment

Tetrachloroethylene has been used widely in the United States as a solvent for domestic and commercial applications. As a result, the chemical can typically be found at low levels (ppb) in ambient air, indoor air, and many public water supplies.

Ambient Air Levels (ug/m^3) (Singh et al, 1983)

City	Average Concentration
Pittsbugh	2.7
Chicago	3.4
Staten Island	2.0
Riverside, CA	3.2
Denver	2.7
St. Louis	2.2
Houston	2.7
Oakland, CA	2.1
Phoenix, AZ	6.7
Los Angeles	10.0

Indoor Air Residential Levels (ug/m^3) (USEPA, 1987b)

Location	Median Concentration
New Jersey Homes (1981)	6.4
New Jersey Homes (1982)	5.5
New Jersey Homes (1983)	6.6
California Homes (1984)	8.3
California Homes (1984)	1.9
California Homes (1984)	1.8

Drinking Water Supplies

Tetrachloroethylene has been found in a number of municipal water supplies throughout the United States. It has been found in finished water of the New Orleans area at up to 5 ug/l (USEPA, 1974) and in eight of ten water utilities surveyed by the USEPA (1975) at levels of 0.07-0.46 ug/l.

Surveys of drinking water supplies in the United States have found that 3% of all public systems derived from well water contain tetrachloroethylene at levels of 0.5 ug/l or higher (USEPA 1987a). A small number of systems (0.7%) have levels higher than 5 ug/l.

G. References

ACGIH, 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. Fifth Edition. Cincinnati, OH: American Conference of Governmental Industrial Hygienists. ISBN: 0-036712-68-6.

ATSDR; December 1988. Toxicological Profile for Tetrachloroethylene; Agency for Toxic Substances and Disease Registry.

Integrated Risk Information System. (IRIS). 1988. Online database.

Singh, H.B., L.J. Salas, R. Stiles, and H. Shigeishi. 1983. Measurements of hazardous organic chemicals in the ambient air. EPA-600/S3-83-002.

U.S. EPA. 1974. Draft analytical report New Orleans Area Water Supply. Surveilance and Analysis Division, Region VI, Dallas, TX. EPA-906/10-74-002.

U.S. EPA. 1975a. Preliminary assessment of suspected carcinogens in drinking water. Report to Congress, Washington, D.C. EPA-56014-75-005 PB.260961.

U.S. EPA (Office of Emergency and Remedial Response). October 1986. Superfund Public Health Evaluation Manual. OSWER Directive, 9285.4-1.

U.S. EPA (Office of Research and Development). July 1985. Health and Assessment Document for Tetrachloroethylene (Perchloroethylene). Washington, DC: EPA/600/8-82/005F.

U.S. EPA (Office of Research and Development). April, 1986. Addendum to the Health and Assessment Document for Tetrachloroethylene (Perchloroethylene). External Review Draft. EPA/600/8-82/005A.

U.S. EPA. 1987a. Tetrachloroethylene (PCE) Health Advisory Office of Drinking Water, U.S. Environmental Protection Agency. March 31, 1987.

U.S. EPA. 1987b. The total exposure assessment (TEAM) methodology. EPA/600/6-86/002.

TETRACHLOROETHYLENE
(Perchloroethylene,Tetrachlorethene, PCE, Perc)

A. Physical and Chemical Properties

Tetrachloroethylene is a halogenated aliphatic hydrocarbon. It is a non-flammable colorless liquid that is soluble in water and common organics. It is widely used as a solvent in degreasing operations and in dry cleaning (4)

Property (a)	Value	Reference
Molecular Weight:	166	US EPA, 1986
Vapor pressure	1.78×10^1 mm Hg	US EPA, 1986
Water solubility	1.5×10^2 mg/l	US EPA, 1986
Henry's law	2.59×10^{-2} atm-m^3/mol	US EPA, 1986
Koc	364 ml/g	US EPA, 1986
log Kow	2.6	US EPA, 1986

(a) Koc = organic carbon-water partition coefficient;
Kow = octanol-water partition coefficient;
Hc = Henry's Law constant.

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiles such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Health Effects

Pharmacokinetics

Tetrachloroethylene is easily absorbed following dermal, gastrointestinal, and inhalation exposures (EPA, 1985). In humans, inhalation is the most common route of exposure. After absorption, tetrachloroethylene is distributed throughout the body. It readily crosses the blood-brain barrier and the placental barrier. The highest concentration can be found in adipose tissue, brain, and the liver. Tetrachloroethylene metabolism is dose-dependent and saturable. Low percentages of the absorbed dose are excreted as metabolites such as trichloroacetic acid or perchloroethylene epoxide. Excretion takes place through the pulmonary route and consists mainly of the unchanged compound (EPA, 1985).

Systemic Effects - Humans

Acute inhalation exposure to low doses of tetrachloroethylene causes symptoms such as dizziness, confusion, nausea, headache, and irritation of the eyes and mucous tissue. At higher concentrations, these symptoms increase and eventually produce faintness and unconsciousness. Chronic exposure enhances the former symptoms. Chronic exposure can also affect short-term memory and produce disorientation, irritability, ataxia, and sleep disturbance. Other reported effects include liver cirrhosis, hepatitis, and nephritis (EPA, 1985).

Systemic Effects - Animals

Effects observed in experimental animals are similar to those reported from human acute and chronic exposures at both low and high concentrations of tetrachloroethylene. The acute oral toxicity has been determined for rats, mice, cats, rabbits, and dogs. No subchronic oral exposure studies exist, and only one chronic study (EPA, 1985).

Carcinogenicity

Tetrachloroethylene is classified as a Group B2/C carcinogen by the EPA. The classification of this substance is currently under review.

There is limited evidence of carcinogenicity in experimental animals from tetrachloroethylene exposure. One study showed a statistically significant increase in the incidence of hepatocellular carcinomas when administered orally to mice. In an inhalation bioassay conducted by the National Toxicology Program, tetrachloroethylene induced a marginally statistically significant increase of mononuclear cell leukemia in male rats and a dose related, but not statistically significant, increase of rare renal tubular cell adenomas and/or carcinomas in male rats (EPA, 1986).

Mutagenicity

Tetrachloroethylene has been studied using a variety of in vivo and in vitro test systems. For the most part, the results reported have been negative. A few positive results have been observed, but problems with purity of the compound and study design flaws make the results difficult to interpret (EPA, 1986).

Reproductive Effects

A developmental study has been conducted in both rats and mice exposed to tetrachloroethylene via inhalation. Tetrachloroethylene has been found to be fetotoxic, but not teratogenic at concentrations that are also maternally toxic. In mice exposed to tetrachloroethylene via inhalation, there is some indication that sperm abnormalities may occur (ATSDR, 1988).

C. Health Criteria

The EPA has developed an oral risk reference dose of 0.01 mg/kg/day. This value is based on the no observed adverse effect level of 14 mg/kg/day in a six week study in mice. At higher doses, liver toxicity was observed. The risk reference dose includes an uncertainty factor of 1000 to account for intraspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent (IRIS, 1988).

The EPA Carcinogen Assessment Group has calculated carcinogenic potency factors for tetrachloroethylene by oral and inhalation exposures as. 5.1×10^{-2} and 1.7×10^{-3} (mg/kg/day)-1, respectively (EPA, 1986).

D. Guidelines and Standards

Water

Ambient Water Quality Criteria: 0.8 ug/1 for 10^{-6} cancer risk level, based on ingesting aquatic organisms and drinking water .

Ambient Water Quality Criteria Adjusted for Drinking Water Only: 0.88 ug/1 for 10^{-6} cancer risk level

MCLG	0 mg/l (proposed)

Air

ACGIH TLV-TWA	50 ppm (335 mg/m^3)
ACGIH STEL	200 ppm (1340 mg/m^3)

E. References

ACGIH, 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. Fifth Edition. Cincinnati, OH: American Conference of Governmental Industrial Hygienists. ISBN: 0-036712-68-6.

U.S. EPA (Office of Emergency and Remedial Response). October 1986. Superfund Public Health Evaluation Manual. OSWER Directive, 9285.4-1.

U.S. EPA (Office of Research and Development). July 1985. Health and Assessment Document for Tetrachloroethylene (Perchloroethylene). Washington, DC: EPA/600/8-82/005F.

U.S. EPA (Office of Research and Development). April, 1986. Addendum to the Health and Assessment Document for Tetrachloroethylene (Perchloroethylene). External Review Draft. EPA/600/8-82/005A.

Integrated Risk Information System. (IRIS). 1988. Online database.

ATSDR; December 1987. Toxicological Profile for Tetrachloroethylene; Agency for Toxic Substances and Disease Registry. Draft for Public Comment.

1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE
(FC-113; Fluorocarbon 113; Freon 113)
CAS #76-13-1

Physical and Chemical Properties

Molecular weight	197.5	ACGIH, 1987
Water solubility	insoluble	ACGIH, 1987
Vapor pressure	284 torr @20°C.	ACGIH, 1987

Uses
FC-113 is used as a dry cleaning solvent and as a refrigerant.

Toxic Effects

Acute exposures to humans:
Based on exposure of human volunteers, the threshold concentration for impairment of psychomotor performance (loss of ability to concentrate, mild lethargy) is about 2500 ppm in the air. No adverse changes in performance of complex mental tasks, clinical status or biochemical tests were observed following exposures to 500 or 1000 ppm for six hours a day, five days/week for two weeks. There were complaints of mild throat irritation on the first day of exposure only (ACGIH, 1987).

Animal studies/Acute exposure:
FC-113 is low in acute toxicity by both the oral and inhalation routes. FC-113 has an oral LD_{50} of 43 g/kg in male rats and an inhalation LC50 for four hours of exposure in rats in the range of 52,000 to 68,000 ppm. High doses resulted in a weak narcotic effect, a potential for cardiac sensitization, irritation of the respiratory tract and liver cell enlargement (ACGIH, 1987).

Undiluted FC-113 produced no significant irritation in a rabbit eye test. However, occluded contact of 5 g/kg with rabibit skin on each of five successive days did result in local necrosis and slogughing, plus conspicuous enlargement of liver cells (ACGIH, 1987).

Animal Studies/Subchronic exposure:
No clinical or pathologic changes developed after 20 six hour exposures of rats, guinea pigs, or dogs to 5100 ppm or 300 seven hour exposures of rats to 2520 ppm.

Carcinogenic effects:
No studies reported.

Mutagenic effects:
An Ames bacterial mutagenic effects test was negative.

Reproductive/Teratogenic effects:
FC-113 caused no changes in the offspring from pregnant rabbits at doses below those causing maternal toxicity.

Guidelines and Standards

ACGIH:	TLV	1000 ppm
	STEL	1250 ppm

References:

American Conference of Governmental Industrial Hygienists (ACGIH). 1987. Documentation of Threshold Limit Values and Biological Exposure Indices. Cincinnati, OH.

BUTYL BENZYL PHTHALATE
(Benzyl butyl phthalate, BBP)

A. Physical and Chemical Properties

Property (a)	Value	Reference
Molecular weight	312 g/mol	Callahan, 1979
Vapor pressure	not available	Callahan, 1979
Log Kow	4.8-5.8	Callahan, 1979

(a) Kow = octanol-water partition coefficient;

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

The US EPA classifies butyl benzyl phthalate as possible human carcinogen, Group C. There is no evidence regarding its carcinogenicity in humans and only limited evidence in animals. The limited evidence includes a statistically significant increase in mononuclear cell leukemia in female rats in a two year feeding study. However, the response in male rats was inconclusive and there was no carcinogenic response in mice in the same feeding study (IRIS, 1988).

In general, mutagenicity tests, including the Salmonella (Ames) test, with butyl benzyl phthalate were negative. Mammalian cytogenicity studies using chinese hampster ovary cells were also negative (IRIS, 1988).

C. References

Callahan, M. et al. 1979. Water Related Environmental Fate of 129 Priority Pollutants. Report to EPA Office of Water Planning and Standards. EPA-440/4-79-029b.

IRIS, 1988. Integrated Risk Information System online computer database maintained by EPA. Searched August, 1988.

BIS(2-ETHYLHEXYL)PHTHALATE
(DEHP, BEHP, di(2-ethylhexyl)phthalate)

A. Physical and Chemical Properties

Bis(2-ethylhexyl)phthalate (BEHP), a phthalic acid ester, is a clear, colorless liquid that is poorly soluble in water but soluble in organic solvents and oils. Phthalic acid esters are primarily used as plasticizers for specific plastics such as polyvinyl chloride. Other uses include as defoaming agents in paper production, perfumes, lubricating oils, and in other industrial and consumer applications (Sittig, 1980).

Property (a)	Value	Reference
Molecular weight	391 g/mol	US EPA, 1986
Vapor pressure	2×10^{-7} mm Hg	US EPA, 1986
Water solubility	0.285 mg/l @24°C.	Verschueren, 1983
Hc	1×10^{-4} atm-m^3/mol	ATSDR, 1988
Log Kow	4.88	ATSDR, 1988

(a) Kow = octanol-water partition coefficient;
Hc = Henry's Law constant

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different data bases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the U.S. EPA.

B. Toxic Effects to Humans

No human data on adverse effects associated with BEHP exposure are available. Several studies on the acute toxic effects of BEHP have been conducted in animals. When administered by oral, intraperitoneal, intravenous, and inhalation routes, BEHP has a low order of acute toxicity. The target organs for high acute exposures are the liver, where morphological and biochemical changes occur, and testes (ATSDR, 1988).

EPA has derived an oral risk reference dose (RfD) of 0.02 mg/kg-day for BEHP. This value is based on a one year study of guinea pigs fed BEHP in their diets. Guinea pigs were the most sensitive species tested. A no observed adverse effect level (NOAEL) was not observed in this study. The lowest observed adverse effect level (LOAEL), 19 mg/kg-day, resulted in an increase in liver weight. An uncertainty factor of 1000 was applied to the LOAEL to obtain the RfD (IRIS, 1988).

In a two-year feeding study of both rats and mice, a statistically significant, dose-related increase in hepatocellular carcinomas and neoplastic nodules was observed in both male and female rats. There is controversy over the adequacy of this study and the significance of these tumors for human risk. Based on this feeding study, EPA derived a carcinogenic potency factor for BEHP of 6.84×10^{-4} $(\text{mg/kg-day})^{-1}$ based on hepatocellular carcinomas in female rats (ATSDR, 1988; USEPA, 1986). The EPA does not provide a carcinogenic potency for BEHP in its IRIS database. ATSDR (1988) and EPA (1986) rank BEHP as a probable human carcinogen, Group B2, based on the EPA Guidelines for Carcinogen Risk Assessment.

Bis(2-ethylhexyl)phthalate is not a mutagenic in most microbial, in vivo or in vitro mammalian test systems. There is no evidence of direct damage to DNA or chromosomes by this chemical (ATSDR, 1988).

Bis(2-ethylhexyl)phthalate has been shown to have adverse effects on fertility and reproduction in both mice and rats. In male mice, the effect is linked to degeneration of seminiferous tubules. DEHP has also been found to be fetotoxic and teratogenic in experimental animals (IRIS, 1988; ATSDR, 1988).

C. Pharmacokinetics

Bis(2-ethylhexyl)phthalate is well absorbed from the gastrointestinal tract via ingestion. It may also be absorbed through the skin and lungs. Hydrolysis to the corresponding monoester metabolite, MEHP, with release of an alcoholic substitute, 2ethylhexanol, largely occurs prior to intestinal absorption. Once absorbed, wide distribution in the body occurs with the liver being the primary storage site. Clearance from the body is rapid and only a slight cumulative potential exists. Bis(2-ethylhexyl)phthalate is converted principally to polar derivatives of the monoesters by oxidative metabolism prior to elimination via urinary excretion. Clearance from the body is rapid, and there is only a slight cumulative potential (ATSDR, 1988).

D. Environmental Effects

The lowest acute effect level for BEHP is 940 ug/l in freshwater and 2944 ug/l in marine environments (IRIS, 1988). In a chronic assay with rainbow trout, BEHP gave a chronic value of 8.4 ug/l. No acute-chronic ration could be calculated because of the absence of a 96 hour LC50 value. In a chronic test with Daphnia magna, significant reproductive impairment was found at 3 ug/l (EPA, 1980).

The biocencentration factors for BEHP with fish and invertebrate species ranged from 54 to 2680 (EPA, 1980).

E. Criteria and Standards

Ambient Water Quality Criteria:
1.5×10^4 µg/l based on ingesting aquatic organisms and drinking water (EPA, 1980)
2.1×10^4 µg/l adjusted for drinking water only (EPA, 1986)

Air:

ACGIH	TLV	5 mg/m^3 8 hr TWA	ACGIH, 1987
	STEL	10 mg/m^3	ACGIH, 1987
OSHA	PEL	5 mg/m^3 TWA	29 CFR 1910

F. References

Agency for Toxic Substances and Disease Registry (ATSDR) 1988. Toxicological Profile for Di(2-ethylhexyl)phthlate. Draft for Public Comment.

American Conference of Governmental Industrial Hygienists (ACGIH) 1987-88. Threshold Limit Values and Biological Exposure Indices. Cincinnati, OH

IRIS, 1988. Integrated Risk Information System online computer database maintained by EPA. Searched August, 1988.

Sittig, M. (ed.) 1980. Priority Toxic Pollutants Health Impacts and Allowable Limits. New Jersey: Noyes Data Corporation pp. 305-310.

U.S. EPA (Office of Emergency and Remedial Response). October 1986. Superfund Public Health Evaluation Manual. EPA 540/1-86/060.

U.S. EPA, 1980. Ambient Water Quality Criteria Document for Phthalate Esters.

Appendix B

APPENDIX B
RISK CALCULATIONS

TABLE B-1
AVERAGE DAILY DOSE AND SITE RISK FROM SOIL INGESTION, DERMAL CONTACT, AND INHALATION

SCENARIO: PHASE I EXPOSED CONSTRUCTION WORKER DURING FOUNDATION CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	6.77E-08	1.43E-09	4.03E-07	8.52E-09	7.19E-08	1.52E-09	0.000047078	0.0000071857	0.0000001144	1.5187028E-11
Benzo(a)anthracene	1.98E-08	4.18E-10	1.18E-07	2.49E-09	9.43E-09	1.99E-10	0.0000137552	0.0000009429	0.0000000048	1.9927313E-12
Benzo(b)fluoranthene	6.69E-08	1.41E-09	3.98E-07	8.42E-09	6.14E-08	1.30E-09	0.0000465195	0.0000061357	0.0000000158	1.2967850E-11
Benzo(k)fluoranthene	6.69E-08	1.41E-09	3.98E-07	8.42E-09	6.14E-08	1.30E-09	0.0000465195	0.0000061357	0.0000000075	1.2967850E-11
Benzo(g,h,i)perylene	1.80E-08	3.81E-10	1.07E-07	2.26E-09	9.43E-09	1.99E-10	0.0000125141	0.0000009429	0.0000000007	1.9927313E-12
Chrysene	1.07E-07	2.25E-09	6.34E-07	1.34E-08	1.05E-07	2.22E-09	0.0000740506	0.0000105214	0.0000000007	2.2237070E-11
Dibenzo(a,h)anthracene	1.65E-08	3.48E-10	9.79E-08	2.07E-09	9.43E-09	1.99E-10	0.0000114385	0.0000009429	0.0000000309	1.9927313E-12
Indeno(1,2,3-c,d)pyrene	1.75E-08	3.70E-10	1.04E-07	2.20E-09	9.43E-09	1.99E-10	0.0000121625	0.0000009429	0.0000000069	1.9927313E-12
Noncarcinogenic PAH Compounds										
Naphthalene(s)	1.91E-08	4.04E-10	1.14E-07	2.40E-09	1.31E-08	2.78E-10	0.0000265589	0.0000026286	0	1.3888734E-12
Fluorene	2.42E-08	5.12E-10	1.44E-07	3.05E-09	1.85E-08	3.91E-10	0.0000168372	0.00000185	0	3.9099804E-12
Anthracene	1.42E-07	2.99E-09	8.43E-07	1.78E-08	1.58E-07	3.35E-09	0.0000984583	0.0000158286	0	3.3453732E-11
Phenanthrene	1.68E-07	3.55E-09	1.00E-06	2.11E-08	1.72E-07	3.64E-09	0.0001167848	0.0000172357	0	3.6427733E-11
Fluoranthene	3.35E-08	7.08E-10	1.99E-07	4.21E-09	9.43E-09	1.99E-10	0.0000232701	0.0000009429	0	1.9927313E-12
Pyrene	1.42E-07	3.01E-09	8.46E-07	1.79E-08	1.52E-07	3.20E-09	0.000098872	0.0000151643	0	3.2049762E-11
Acenaphthene	2.56E-08	5.42E-10	1.52E-07	3.22E-09	2.06E-08	4.36E-10	0.0000178094	0.0000020643	0	4.3628739E-12
Pesticides										
Aldrin	2.47E-09	5.22E-11	1.47E-08	3.10E-10	2.70E-09	5.71E-11	0.0005718122	0.00009	0.0000000062	1.7119374E-15
Dieldrin	2.38E-10	5.03E-12	1.42E-09	2.99E-11	1.43E-10	3.02E-12	0.0000330952	0.0000028571	0.0000000006	1.5096450E-16
Heptachlor	1.40E-09	2.96E-11	8.33E-09	1.76E-10	1.43E-10	3.02E-12	0.0000324517	0.0000004762	0.0000000009	9.0578697E-16
Heptachlor Epoxide	3.54E-10	7.48E-12	2.11E-09	4.45E-11	1.43E-10	3.02E-12	0.0001892227	0.000010989	0.0000000005	3.9250769E-17
DDTR*	2.64E-09	5.59E-11	1.57E-08	3.33E-10	2.81E-09	5.95E-11	0.0000367541	0.0000056286	1.3205583E-10	2.9740006E-14
Endosulfan I	5.29E-10	1.12E-11	3.15E-09	6.65E-11	2.86E-10	6.04E-12	0.000073545	0.0000057143	0	3.0192899E-16
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	3.13E-09	6.61E-11	1.86E-08	3.93E-10	2.86E-10	6.04E-12	0.0004346508	0.0000057143	0	3.0192899E-16
Alpha-BHC	6.06E-10	1.28E-11	3.60E-09	7.62E-11	1.43E-10	3.02E-12	0.0000140348	0.0000004762	0.0000000006	9.0578697E-16
Beta-BHC	1.04E-09	2.20E-11	6.19E-09	1.31E-10	1.43E-10	3.02E-12	0.000024086	0.0000004762	0.0000000003	9.0578697E-16
Gamma-BHC	2.45E-10	5.17E-12	1.46E-09	3.08E-11	1.43E-10	3.02E-12	0.0000056691	0.0000004762	4.6728404E-11	9.0578697E-16
Methyoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	4.26E-08	9.00E-10	2.53E-07	5.35E-09	4.13E-08	8.72E-10	0.0000147936	0.0000020635	8.7545519E-11	1.7444786E-11
Di-N-octyl phthalate	5.60E-08	1.18E-09	3.33E-07	7.04E-09	5.71E-08	1.21E-09	0.00001946	0.0000028571	0	2.4154319E-11
Di-N-butyl phthalate	3.04E-08	6.43E-10	1.81E-07	3.83E-09	2.86E-08	6.04E-10	0.0000021148	0.0000002857	0	6.0385798E-11
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE 8-1
AVERAGE DAILY DOSE AND SITE RISK FROM SOIL INGESTION, DERMAL CONTACT, AND INHALATION

SCENARIO: PHASE I EXPOSED CONSTRUCTION WORKER DURING FOUNDATION CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	1.33E-09	2.80E-11	2.37E-08	5.00E-10	0.00E+00	0.00E+00				
Methylene Chloride	4.77E-10	1.01E-11	8.51E-09	1.80E-10	0.00E+00	0.00E+00	0.0000001499	0	1.4252118E-12	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	1.78E-09	3.76E-11	3.17E-08	6.70E-10	0.00E+00	0.00E+00	0.000008374	0	4.0352701E-11	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	2.18E-08	4.61E-10	3.89E-07	8.23E-09	0.00E+00	0.00E+00	0.0000041126	0	0	0
Trichlorofluoromethane	1.08E-09	2.28E-11	1.93E-08	4.07E-10	0.00E+00	0.00E+00				
Xylenes	7.67E-08	1.62E-09	1.37E-06	2.89E-08	0.00E+00	0.00E+00	0.000000723	0	0	0
Metals										
Arsenic	1.58E-06	3.34E-08	0.00E+00	0.00E+00	2.54E-07	5.37E-09	0.0015802988	0.0002542857	0.0000000501	5.3743360E-12
Lead	2.92E-06	6.17E-08	0.00E+00	0.00E+00	2.57E-06	5.43E-08	0.0058345238	0.0051428571	0	2.7173609E-11
Mercury	5.10E-08	1.08E-09	0.00E+00	0.00E+00	1.06E-08	2.23E-10	0.0001700337	0.0000352381	0	6.7028236E-14
Zinc	9.96E-06	2.11E-07	0.00E+00	0.00E+00	2.11E-06	4.47E-08	0.0000474446	0.0002114286	0	0.0000000004
Copper	5.75E-06	1.21E-07	0.00E+00	0.00E+00	7.07E-07	1.49E-08	0.0001552828	0.0000707143	0	1.4945485E-10
Silver	1.81E-07	3.82E-09	0.00E+00	0.00E+00	7.14E-09	1.51E-10	0.0000602797	0.000002381	0	4.5289349E-13
Thallium	1.28E-06	2.71E-08	0.00E+00	0.00E+00	2.50E-07	5.28E-09	0.0183106576	0.0035714286	0	3.6986301E-13
TOTAL SITE HAZARD INDEX								0.0377920112		
TOTAL SITE CANCER RISK										0.000000242

TABLE B-2
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND INHALATION

SCENARIO: EXPOSED CONSTRUCTION WORKERS DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INAHALATION (MG/KG/DAY)	ADD(LIFE) INAHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	7.34E-07	1.55E-08	8.73E-06	1.85E-07	8.81E-07	1.86E-08	0.0009467679	0.0000880714	0.0000023011	1.8613922E-10
Benzo(a)anthracene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000179	9.9636567E-12
Benzo(b)fluoranthene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000172	9.9636567E-12
Benzo(k)fluoranthene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000081	9.9636567E-12
Benzo(g,h,i)perylene	3.93E-08	8.30E-10	4.67E 07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000027	9.9636567E-12
Chrysene	1.31E-06	2.77E-08	1.56E-05	3.29E-07	1.57E-06	3.32E-08	0.0016892857	0.0001571429	0.0000000164	0.0000000003
Dibenzo(a,h)anthracene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000001367	9.9636567E-12
Indeno(1,2,3-c,d)pyrene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000286	9.9636567E-12
Noncarcinogenic PAH Compounds										
Naphthalene(s)	3.65E-07	7.71E-09	4.34E-06	9.18E-08	4.44E-07	9.39E-09	0.0009413929	0.0000888889	0	4.6966732E-11
Fluorene	4.01E-07	8.47E-09	4.77E-06	1.01E-07	4.81E-07	1.02E-08	0.0005167679	0.0000480714	0	1.0159911E-10
Anthracene	4.48E-06	9.48E-08	5.34E-05	1.13E-06	5.38E-06	1.14E-07	0.0057842679	0.0005380714	0	0.0000000011
Phenanthrene	4.64E-06	9.80E-08	5.52E-05	1.17E-06	5.56E-06	1.18E-07	0.0059816071	0.0005564286	0	0.0000000012
Fluoranthene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0	9.9636567E-12
Pyrene	3.16E-06	6.68E-08	3.76E-05	7.95E-07	3.79E-06	8.02E-08	0.0040773214	0.0003792857	0	0.0000000008
Acenaphthene	3.89E-07	8.21E-09	4.63E-06	9.78E-08	4.66E-07	9.86E-09	0.0005014107	0.0000466429	0	9.8579815E-11
Pesticides										
Aldrin	2.26E-08	4.77E-10	2.68E-07	5.67E-09	2.71E-08	5.72E-10	0.0097005952	0.000902381	0.0000001046	1.7164663E-14
Dieldrin	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0001535714	0.0000142857	0.0000000026	7.5482248E-16
Heptachlor	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	0.0000000007	4.5289349E-15
Heptachlor Epoxide	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0005906593	0.0000549451	0.0000000015	3.9625384E-16
DDTR*	1.19E-09	2.52E-11	1.42E-08	2.99E-10	1.43E-09	3.02E-11	0.0000307143	0.0000028571	1.1035505E-10	1.5096450E-14
Endosulfan I	4.58E-09	9.69E-11	5.45E-08	1.15E-09	5.50E-09	1.16E-10	0.0011825	0.00011	0	5.8121331E-15
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	1.19E-09	2.52E-11	1.42E-08	2.99E-10	1.43E-09	3.02E-11	0.0003071429	0.0000285714	0	1.5096450E-15
Alpha-BHC	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	0.000000001	4.5289349E-15
Beta-BHC	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	0.0000000003	4.5289349E-15
Gamma-BHC	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	2.1097288E-10	4.5289349E-15
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	9.70E-08	2.05E-09	1.15E-06	2.44E-08	1.17E-07	2.46E-09	0.0000625804	0.0000058254	0.0000000004	4.9247973E-11
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-2
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND INHALATION

SCENARIO: EXPOSED CONSTRUCTION WORKERS DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	2.66E-06	5.62E-08	0.00E+00	0.00E+00	9.57E-07	2.02E-08	0.0026587302	0.0009571429	0.0000000843	2.0229242E-11
Lead	1.07E-05	2.25E-07	0.00E+00	0.00E+00	1.28E-05	2.70E-07	0.0213095238	0.0255714286	0	1.3511322E-10
Mercury	1.65E-07	3.48E-09	0.00E+00	0.00E+00	5.93E-08	1.25E-09	0.0005489418	0.000197619	0	3.7590159E-13
Zinc	3.17E-05	6.71E-07	0.00E+00	0.00E+00	1.14E-05	2.42E-07	0.0001611716	0.0011428571	0	0.0000000024
Copper	8.53E-06	1.80E-07	0.00E+00	0.00E+00	3.07E-06	6.49E-08	0.0002305877	0.0003071429	0	0.0000000006
Silver	9.92E-08	2.10E-09	0.00E+00	0.00E+00	3.57E-08	7.55E-10	0.0000330688	0.0000119048	0	2.2644674E-12
Thallium	3.47E-06	7.34E-08	0.00E+00	0.00E+00	1.25E-06	2.64E-08	0.0496031746	0.0178571429	0	1.8493151E-12
TOTAL SITE HAZARD INDEX								0.1565681451		
TOTAL SITE CANCER RISK										2.73E-06

TABLE B-3
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE I EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INAHALATION (MG/KG/DAY)	ADD(LIFE) INAHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.17E-09	1.94E-10	0	0.0000009174	0	1.9389502E-12
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Benzo(b)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Benzo(k)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.64E-08	3.46E-10	0	0.0000016369	0	3.4596030E-12
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Noncarcinogenic PAH Compounds										
Naphthalene(s)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.56E-09	9.64E-11	0	0.0000009122	0	4.8198560E-13
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.01E-09	1.06E-10	0	0.0000005007	0	1.0583240E-12
Anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.60E-08	1.18E-09	0	0.0000056049	0	1.1845995E-11
Phenanthrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.80E-08	1.23E-09	0	0.0000057961	0	1.2250140E-11
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.95E-08	8.35E-10	0	0.0000039509	0	8.3502237E-12
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.86E-09	1.03E-10	0	0.0000004859	0	1.0268731E-12
Pesticides										
Aldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.82E-10	5.96E-12	0	0.0000093998	0	1.7879857E-16
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000001488	0	7.8627341E-18
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000005723	0	2.0443109E-18
DDTR*	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.49E-11	3.15E-13	0	0.0000000298	0	1.5725468E-16
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.73E-11	1.21E-12	0	0.0000011458	0	6.0543053E-17
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.49E-11	3.15E-13	0	0.0000002976	0	1.5725468E-17
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Methyoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.21E-09	2.56E-11	0	0.0000000607	0	5.1299972E-13
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-3
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE I EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.97E-09	2.11E-10	0	0.0000099702	0	2.1072127E-13
Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-07	2.81E-09	0	0.000266369	0	1.4074294E-12
Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.18E-10	1.31E-11	0	0.0000020585	0	3.9156416E-15
Zinc	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.19E-07	2.52E-09	0	0.0000119048	0	2.5160749E-11
Copper	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.20E-08	6.76E-10	0	0.0000031994	0	6.7619514E-12
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.72E-10	7.86E-12	0	0.000000124	0	2.3588202E-14
Thallium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.30E-08	2.75E-10	0	0.0001860119	0	1.9263699E-14
TOTAL SITE HAZARD INDEX								0.0005115408		
TOTAL SITE CANCER RISK										7.5239841E-11

TABLE 6-4
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE I EXPOSED OFF SITE CHILDREN DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.57E-08	1.18E-09	0	0.0000055656	0	1.1762965E-11
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Benzo(b)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Benzo(k)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.93E-08	2.10E-09	0	0.0000099306	0	2.0988258E-11
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Noncarcinogenic PAH Compounds										
Naphthalene(s)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.77E-08	5.85E-10	0	0.000005534	0	2.9240460E-12
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.04E-08	6.42E-10	0	0.0000030378	0	6.4204990E-12
Anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.40E-07	7.19E-09	0	0.0000340031	0	7.1865705E-11
Phenanthrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.52E-07	7.43E-09	0	0.0000351632	0	7.4317515E-11
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.98E-09	6.30E-11	0	0.0000002979	0	6.2964775E-13
Pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.40E-07	5.07E-09	0	0.0000239688	0	5.0658023E-11
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.95E-08	6.23E-10	0	0.0000029476	0	6.2296967E-12
Pesticides										
Aldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-09	3.62E-11	0	0.0000570255	0	1.0847114E-15
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.51E-11	9.54E-13	0	0.0000009028	0	4.7700587E-17
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.51E-11	9.54E-13	0	0.0000001505	0	2.8620352E-16
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.51E-11	9.54E-13	0	0.0000034722	0	1.2402153E-17
DDTR*	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.03E-11	1.91E-12	0	0.0000001806	0	9.5401174E-16
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.48E-10	7.35E-12	0	0.0000069514	0	3.6729452E-16
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.03E-11	1.91E-12	0	0.0000018056	0	9.5401174E-17
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.51E-11	9.54E-13	0	0.0000001505	0	2.8620352E-16
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.51E-11	9.54E-13	0	0.0000001505	0	2.8620352E-16
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.51E-11	9.54E-13	0	0.0000001505	0	2.8620352E-16
Methyoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.36E-09	1.56E-10	0	0.0000003681	0	3.1115460E-12
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-4
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE I EXPOSED OFF SITE CHILDREN DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INAHALATION (MG/KG/DAY)	ADD(LIFE) INAHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.05E-08	1.28E-09	0	0.0000604861	0	1.2783757E-12
Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.08E-07	1.71E-08	0	0.0016159722	0	8.5384051E-12
Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.75E-09	7.92E-11	0	0.0000124884	0	2.3754892E-14
Zinc	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.22E-07	1.53E-08	0	0.0000722222	0	1.5264188E-10
Copper	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.94E-07	4.10E-09	0	0.0000194097	0	4.1022505E-11
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.26E-09	4.77E-11	0	0.0000007523	0	1.4310176E-13
Thallium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.90E-08	1.67E-09	0	0.0011284722	0	1.1686644E-13
TOTAL SITE HAZARD INDEX								0.0031033472		
TOTAL SITE CANCER RISK										4.56E-10

TABLE B-5
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND DERMAL CONTACT

SCENARIO: PHASE II EXPOSED CONSTRUCTION WORKERS DURING FOUNDATION CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INAHALATION (MG/KG/DAY)	ADD(LIFE) INAHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	1.56E-07	3.31E-09	9.31E-07	1.97E-08	0.00E+00	0.00E+00	0.0001087179	0	0.0000002642	0
Benzo(a)anthracene	1.96E-08	4.15E-10	1.17E-07	2.47E-09	0.00E+00	0.00E+00	0.0000136518	0	0.0000000048	0
Benzo(b)fluoranthene	1.96E-08	4.15E-10	1.17E-07	2.47E-09	0.00E+00	0.00E+00	0.0000136518	0	0.0000000046	0
Benzo(k)fluoranthene	1.96E-08	4.15E-10	1.17E-07	2.47E-09	0.00E+00	0.00E+00	0.0000136518	0	0.0000000022	0
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chrysene	2.71E-07	5.73E-09	1.61E-06	3.41E-08	0.00E+00	0.00E+00	0.0001883946	0	0.0000000018	0
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	8.08E-08	1.71E-09	4.81E-07	1.02E-08	0.00E+00	0.00E+00	0.0001123583	0	0	0
Fluorene	9.43E-08	1.99E-09	5.61E-07	1.19E-08	0.00E+00	0.00E+00	0.0000655286	0	0	0
Anthracene	9.05E-07	1.91E-08	5.38E-06	1.14E-07	0.00E+00	0.00E+00	0.0006287268	0	0	0
Phenanthrene	9.53E-07	2.01E-08	5.67E-06	1.20E-07	0.00E+00	0.00E+00	0.000662153	0	0	0
Fluoranthene	2.04E-08	4.30E-10	1.21E-07	2.56E-09	0.00E+00	0.00E+00	0.0000141482	0	0	0
Pyrene	6.44E-07	1.36E-08	3.83E-06	8.10E-08	0.00E+00	0.00E+00	0.0004475304	0	0	0
Acenaphthene	8.94E-08	1.89E-09	5.32E-07	1.12E-08	0.00E+00	0.00E+00	0.0000621363	0	0	0
Pesticides										
Aldrin	4.63E-09	9.79E-11	2.76E-08	5.82E-10	0.00E+00	0.00E+00	0.0010728373	0	0.0000000116	0
Dieldrin	4.46E-10	9.44E-12	2.66E-09	5.61E-11	0.00E+00	0.00E+00	0.0000620536	0	0.000000001	0
Heptachlor	6.04E-10	1.28E-11	3.59E-09	7.60E-11	0.00E+00	0.00E+00	0.0000139965	0	0.0000000004	0
Heptachlor Epoxide	4.61E-10	9.75E-12	2.74E-09	5.80E-11	0.00E+00	0.00E+00	0.0002466232	0	0.0000000006	0
DDTR*	5.86E-10	1.24E-11	3.49E-09	7.37E-11	0.00E+00	0.00E+00	0.0000081497	0	2.9281514E-11	0
Endosulfan I	1.37E-09	2.89E-11	8.13E-09	1.72E-10	0.00E+00	0.00E+00	0.0001898839	0	0	0
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	3.57E-10	7.55E-12	2.13E-09	4.49E-11	0.00E+00	0.00E+00	0.0000496429	0	0	0
Alpha-BHC	5.60E-10	1.18E-11	3.33E-09	7.04E-11	0.00E+00	0.00E+00	0.0000129623	0	0.0000000005	0
Beta-BHC	3.29E-09	6.96E-11	1.96E-08	4.14E-10	0.00E+00	0.00E+00	0.0000763259	0	0.0000000009	0
Gamma-BHC	3.69E-10	7.80E-12	2.20E-09	4.64E-11	0.00E+00	0.00E+00	0.0000085496	0	7.0471484E-11	0
Methyoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	2.73E-08	5.77E-10	1.62E-07	3.43E-09	0.00E+00	0.00E+00	0.0000094818	0	5.6111389E-11	0
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compouods										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE 8-5
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND DERMAL CONTACT

SCENARIO: PHASE II EXPOSED CONSTRUCTION WORKERS DURING FOUNDATION CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INAHALATION (MG/KG/DAY)	ADD(LIFE) INAHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	8.81E-07	1.86E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0008809524	0	0.0000000279	0
Lead	3.08E-06	6.52E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0061666667	0	0	0
Mercury	4.04E-08	8.54E-10	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0001346561	0	0	0
Zinc	1.13E-05	2.38E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000536659	0	0	0
Copper	3.77E-06	7.97E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0001018876	0	0	0
Silver	6.75E-08	1.43E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000224868	0	0	0
Thallium	1.25E-06	2.64E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0178571429	0	0	0
TOTAL SITE HAZARD INDEX								0.0292986143		
TOTAL SITE CANCER RISK										3.21E-07

TABLE 8-6
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND INHALATION

SCENARIO: PHASE II EXPOSED CONSTRUCTION WORKERS DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	1.43E-07	3.01E-09	1.70E-06	3.59E-08	1.71E-07	3.62E-09	0.0001839786	0.0000171143	0.0000004472	3.6171093E-11
Benzo(a)anthracene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000179	9.9636567E-12
Benzo(b)fluoranthene	1.69E-07	3.57E-09	2.01E-06	4.25E-08	2.03E-07	4.28E-09	0.0002177643	0.0000202571	0.0000000741	4.2813531E-11
Benzo(k)fluoranthene	1.69E-07	3.57E-09	2.01E-06	4.25E-08	2.03E-07	4.28E-09	0.0002177643	0.0000202571	0.0000000349	4.2813531E-11
Benzo(g,h,i)perylene	3.97E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0.0000000027	9.9636567E-12
Chrysene	2.77E-07	5.85E-09	3.30E-06	6.96E-08	3.32E-07	7.02E-09	0.0003572071	0.0000332286	0.0000000035	7.0228683E-11
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0001013571	0.0000094286	0	4.9818283E-12
Fluorene	6.95E-08	1.47E-09	8.27E-07	1.75E-08	8.34E-08	1.76E-09	0.0000896857	0.0000083429	0	1.7632653E-11
Anthracene	6.10E-07	1.29E-08	7.26E-06	1.53E-07	7.32E-07	1.55E-08	0.0007872071	0.0000732286	0	1.5476880E-10
Phenanthrene	6.10E-07	1.29E-08	7.26E-06	1.53E-07	7.32E-07	1.55E-08	0.0007872071	0.0000732286	0	1.5476880E-10
Fluoranthene	3.93E-08	8.30E-10	4.67E-07	9.88E-09	4.71E-08	9.96E-10	0.0000506786	0.0000047143	0	9.9636567E-12
Pyrene	4.77E-07	1.01E-08	5.68E-06	1.20E-07	5.72E-07	1.21E-08	0.0006152071	0.0000572286	0	1.2095275E-10
Acenaphthene	6.24E-08	1.32E-09	7.42E-07	1.57E-08	7.49E-08	1.58E-09	0.0000804714	0.0000074857	0	1.5821079E-11
Pesticides										
Aldrin	9.83E-09	2.08E-10	1.17E-07	2.47E-09	1.18E-08	2.49E-10	0.0042283333	0.0003933333	0.0000000456	7.4818004E-15
Dieldrin	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0001535714	0.0000142857	0.0000000026	7.5482248E-16
Heptachlor	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	0.0000000007	4.5289349E-15
Heptachlor Epoxide	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0005906593	0.0000549451	0.0000000015	1.9625384E-16
DDTR*	4.62E-09	9.76E-11	5.50E-08	1.16E-03	5.54E-09	1.17E-10	0.0001191714	0.0000110857	0.0000000004	5.8574224E-14
Endosulfan I	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0001535714	0.0000142857	0	7.5482248E-16
Endosulfan II	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0001535714	0.0000142857	0	7.5482248E-16
Endosulfan sulfate	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0001535714	0.0000142857	0	7.5482248E-16
Alpha-BHC	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	0.000000001	1.5289349E-15
Beta-BHC	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	0.0000000003	4.5289349E-15
Gamma-BHC	5.95E-10	1.26E-11	7.08E-09	1.50E-10	7.14E-10	1.51E-11	0.0000255952	0.000002381	2.1097288E-10	4.5289349E-15
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors	1.67E-08	3.53E-10	1.99E-07	4.20E-09	0.00E+00	0.00E+00	0.0021561429	0	0.0000000351	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	6.43E-09	1.36E-10	7.65E-08	1.62E-09	7.94E-08	1.68E-09	0.0000041464	0.0000039683	0	3.3547666E-11
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	3.73E-06	7.88E-08	0.00E+00	0.00E+00	1.34E-06	2.84E-08	0.0037301587	0.0013428571	0.0000001183	2.8381325E-11
Lead	1.37E-05	2.89E-07	0.00E+00	0.00E+00	1.64E-05	3.47E-07	0.0273809524	0.0328571429	0	1.7360917E-10
Mercury	1.98E-08	4.19E-10	0.00E+00	0.00E+00	7.14E-09	1.51E-10	0.0000661376	0.0000238095	0	4.5289349E-14
Zinc	4.68E-05	9.90E-07	0.00E+00	0.00E+00	1.69E-05	3.56E-07	0.0002229781	0.0016857143	0	0.0000000036
Copper	9.92E-06	2.10E-07	0.00E+00	0.00E+00	3.57E-06	7.55E-08	0.0002681253	0.0003571429	0	0.0000000008
Silver	5.16E-07	1.09E-08	0.00E+00	0.00E+00	1.86E-07	3.93E-09	0.0001719577	0.0000619048	0	1.1775231E-11
Thallium	3.85E-06	8.14E-08	0.00E+00	0.00E+00	1.39E-06	2.93E-08	0.0549886621	0.0197959184	0	2.0500978E-12
TOTAL SITE HAZARD INDEX								0.1552224093		
TOTAL SITE CANCER RISK										7.91E-07

TABLE B-7
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE II EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-09	3.77E-11	0	0.0000001783	0	3.7678222E-13
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Benzo(b)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.11E-09	4.46E-11	0	0.000000211	0	4.4597428E-13
Benzo(k)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.11E-09	4.46E-11	0	0.000000211	0	4.4597428E-13
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.46E-09	7.32E-11	0	0.0000003461	0	7.3154878E-13
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000982	0	5.1894045E-14
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.69E-10	1.84E-11	0	0.0000000869	0	1.8367347E-13
Anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-09	1.61E-10	0	0.0000007628	0	1.6121750E-12
Phenanthrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-09	1.61E-10	0	0.0000007628	0	1.6121750E-12
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	1.04E-11	0	0.0000000491	0	1.0378809E-13
Pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.96E-09	1.26E-10	0	0.0000005961	0	1.2599245E-12
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.80E-10	1.65E-11	0	0.000000078	0	1.6480291E-13
Pesticides										
Aldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-10	2.60E-12	0	0.0000040972	0	7.7935421E-17
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000001488	0	7.8627341E-18
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000005723	0	2.0443109E-18
DDTR*	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.77E-11	1.22E-12	0	0.0000001155	0	6.1014817E-16
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000001488	0	7.8627341E-18
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000001488	0	7.8627341E-18
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000001488	0	7.8627341E-18
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	1.57E-13	0	0.0000000248	0	4.7176405E-17
Methyoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.27E-10	1.75E-11	0	0.0000000413	0	3.4945485E-13
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-8
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE II EXPOSED OFF SITE CHILDREN DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-08	3.52E-10	0	0.0000016639	0	3.5166341E-12
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	9.69E-11	0	0.0000004583	0	9.6868885E-13
Benzo(b)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-08	4.16E-10	0	0.0000019694	0	4.1624266E-12
Benzo(k)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-08	4.16E-10	0	0.0000019694	0	4.1624266E-12
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	9.69E-11	0	0.0000004583	0	9.6868885E-13
Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.23E-08	6.83E-10	0	0.0000032306	0	6.8277886E-12
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	9.69E-11	0	0.0000009167	0	4.8434442E-13
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.11E-09	1.71E-10	0	0.0000008111	0	1.7142857E-12
Anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.12E-08	1.50E-09	0	0.0000071194	0	1.5046967E-11
Phenanthrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.12E-08	1.50E-09	0	0.0000071194	0	1.5046967E-11
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	9.69E-11	0	0.0000004583	0	9.6868885E-13
Pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.56E-08	1.18E-09	0	0.0000055639	0	1.1759295E-11
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.28E-09	1.54E-10	0	0.0000007278	0	1.5381605E-12
Pesticides										
Aldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.15E-09	2.42E-11	0	0.0000382407	0	7.2739726E-16
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000013889	0	7.3385519E-17
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000002315	0	4.4031311E-16
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000053419	0	1.9080235E-17
DDTR*	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.39E-10	1.14E-11	0	0.0000010778	0	5.6947162E-15
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000013889	0	7.3385519E-17
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000013889	0	7.3385519E-17
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000013889	0	7.3385519E-17
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000002315	0	4.4031311E-16
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000002315	0	4.4031311E-16
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	1.47E-12	0	0.0000002315	0	4.4031311E-16
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.01E-09	1.48E-10	0	0.0000003507	0	2.9647750E-12
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-7
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE II EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-08	2.96E-10	0	0.0000139881	0	2.9563880E-13
Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-07	3.62E-09	0	0.0003422619	0	1.8084289E-12
Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-11	1.57E-12	0	0.000000248	0	4.7176405E-16
Zinc	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.76E-07	3.71E-09	0	0.0000175595	0	3.7112105E-11
Copper	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.72E-08	7.86E-10	0	0.0000037202	0	7.8627341E-12
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.93E-09	4.09E-11	0	0.0000006448	0	1.2265865E-13
Thallium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-08	3.05E-10	0	0.0002062075	0	2.1355186E-14
TOTAL SITE HAZARD INDEX								0.0005936295		
TOTAL SITE CANCER RISK										5.4770046E-11

TABLE 8-8
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE II EXPOSED OFF SITE CHILDREN DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KC/DAY)	ADD(CHRONIC) INAHALATION (MG/KG/DAY)	ADD(LIFE) INAHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-07	2.76E-09	0	0.0001305556	0	2.7592955E-12
Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.60E-06	3.38E-08	0	0.0031944444	0	1.6878669E-11
Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-10	1.47E-11	0	0.0000023148	0	4.4031311E-15
Zinc	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.64E-06	3.46E-08	0	0.0001638889	0	0.0000000003
Copper	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.47E-07	7.34E-09	0	0.0000347222	0	7.3385519E-11
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-08	3.82E-10	0	0.0000060185	0	1.1448141E-12
Thallium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-07	2.85E-09	0	0.0019246032	0	1.9931507E-13
TOTAL SITE HAZARD INDEX								0.0055405069		
TOTAL SITE CANCER RISK										5.11E-10

TABLE 8-9
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND DERMAL CONTACT

SCENARIO: PHASE III EXPOSED CONSTRUCTION WORKERS DURING FOUNDATION EXCAVATION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	3.64E-08	1.54E-09	2.16E-07	9.15E-09	0.00E+00	0.00E+00	0.0000252848	0	0.0000001229	0
Benzo(a)anthracene	1.57E-08	6.64E-10	9.35E-08	3.95E-09	0.00E+00	0.00E+00	0.0000109214	0	0.0000000077	0
Benzo(b)fluoranthene	4.16E-08	1.76E-09	2.48E-07	1.05E-08	0.00E+00	0.00E+00	0.0000289252	0	0.0000000197	0
Benzo(k)fluoranthene	4.16E-08	1.76E-09	2.48E-07	1.05E-08	0.00E+00	0.00E+00	0.0000289252	0	0.0000000093	0
Benzo(g,h,i)perylene	1.57E-08	6.64E-10	9.35E-08	3.95E-09	0.00E+00	0.00E+00	0.0000109214	0	0.0000000012	0
Chrysene	6.52E-08	2.76E-09	3.88E-07	1.64E-08	0.00E+00	0.00E+00	0.0000453157	0	0.0000000009	0
Dibenzo(a,h)anthracene	7.86E-09	3.32E-10	4.67E-08	1.98E-09	0.00E+00	0.00E+00	0.0000054607	0	0.0000000295	0
Indeno(1,2,3-c,d)pyrene	9.82E-09	4.15E-10	5.84E-08	2.47E-09	0.00E+00	0.00E+00	0.0000068259	0	0.0000000077	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	9.29E-08	3.93E-09	5.53E-07	2.34E-08	0.00E+00	0.00E+00	0.0001291542	0	0	0
Fluorene	2.37E-08	1.00E-09	1.41E-07	5.97E-09	0.00E+00	0.00E+00	0.0000184897	0	0	0
Anthracene	1.30E-07	5.49E-09	7.73E-07	3.27E-08	0.00E+00	0.00E+00	0.0000902838	0	0	0
Phenanthrene	1.34E-07	5.64E-09	7.95E-07	3.36E-08	0.00E+00	0.00E+00	0.0000928073	0	0	0
Fluoranthene	1.77E-08	7.47E-10	1.05E-07	4.45E-09	0.00E+00	0.00E+00	0.0000122866	0	0	0
Pyrene	1.05E-07	4.45E-09	6.26E-07	2.65E-08	0.00E+00	0.00E+00	0.0000731157	0	0	0
Acenaphthene	2.03E-08	8.59E-10	1.21E-07	5.11E-09	0.00E+00	0.00E+00	0.0000141317	0	0	0
Pesticides										
Aldrin	2.41E-09	1.02E-10	1.43E-08	6.06E-10	0.00E+00	0.00E+00	0.0005584069	0	0.000000012	0
Dieldrin	4.27E-10	1.81E-11	2.54E-09	1.08E-10	0.00E+00	0.00E+00	0.000059421	0	0.000000002	0
Heptachlor	1.17E-09	4.95E-11	6.97E-09	2.95E-10	0.00E+00	0.00E+00	0.0000271281	0	0.0000000015	0
Heptachlor Epoxide	3.73E-10	1.58E-11	2.22E-09	9.39E-11	0.00E+00	0.00E+00	0.0001996129	0	0.000000001	0
DDTRs	1.09E-09	4.61E-11	6.49E-09	2.75E-10	0.00E+00	0.00E+00	0.0000151727	0	1.0902939E-10	0
Endosulfan I	4.00E-10	1.69E-11	2.38E-09	1.01E-10	0.00E+00	0.00E+00	0.0000556602	0	0	0
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Alpha-BHC	1.14E-09	4.80E-11	6.76E-09	2.86E-10	0.00E+00	0.00E+00	0.0000263258	0	0.0000000021	0
Beta-BHC	2.62E-10	1.11E-11	1.56E-09	6.59E-11	0.00E+00	0.00E+00	0.0000060675	0	1.3849483E-10	0
Gamma-BHC	6.41E-10	2.71E-11	3.81E-09	1.61E-10	0.00E+00	0.00E+00	0.0000148427	0	0.0000000002	0
Methyoxychlor	6.29E-10	2.66E-11	3.74E-09	1.58E-10	0.00E+00	0.00E+00	0.0000000874	0	0	0
Total Arochlore	5.72E-09	2.42E-10	3.41E-08	1.44E-09	0.00E+00	0.00E+00	0.0003978048	0	0.0000000129	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	2.03E-08	8.58E-10	1.21E-07	5.10E-09	0.00E+00	0.00E+00	0.0000070534	0	8.3481605E-11	0
Di-N-octyl phthalate	1.99E-08	8.39E-10	1.18E-07	4.99E-09	0.00E+00	0.00E+00	0.0000069004	0	0	0
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	3.03E-10	1.28E-11	5.41E-09	2.29E-10	0.00E+00	0.00E+00	0.0000081569	0	1.2550506E-11	0
Chlorobenzene	4.68E-09	1.98E-10	8.36E-08	3.53E-09	0.00E+00	0.00E+00	0.0000176532	0	0	0

TABLE B-9
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND DERMAL CONTACT

SCENARIO: PHASE III EXPOSED CONSTRUCTION WORKERS DURING FOUNDATION EXCAVATION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	5.78E-10	2.44E-11	1.03E-08	4.36E-10	0.00E+00	0.00E+00	0.0000017119	0	2.6231961E-11	0
1,1,2,2 Tetrachloroethane	7.14E-10	3.02E-11	1.28E-08	5.39E-10	0.00E+00	0.00E+00				
Toluene	9.52E-10	4.03E-11	1.70E-08	7.19E-10	0.00E+00	0.00E+00	0.0000000598	0	0	0
Ethylbenzene	9.18E-08	3.88E-09	1.64E-06	6.93E-08	0.00E+00	0.00E+00	0.0000173131	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	1.56E-07	6.58E-09	2.78E-06	1.17E-07	0.00E+00	0.00E+00	0.0000014663	0	0	0
Metals										
Arsenic	1.34E-06	5.67E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0013418367	0	0.0000000851	0
Lead	5.39E-06	2.28E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0107813187	0	0	0
Mercury	3.56E-08	1.50E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0001186697	0	0	0
Zinc	1.17E-05	4.97E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000559335	0	0	0
Copper	3.33E-06	1.41E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000900901	0	0	0
Silver	2.18E-07	9.20E-09	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0000725624	0	0	0
Thallium	1.32E-06	5.56E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0107894678	0	0	0
TOTAL SITE HAZARD INDEX								0.033261583		
TOTAL SITE CANCER RISK										3.16E-07

TABLE 6-10

AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND INHALATION

SCENARIO: PHASE II EXPOSED CONSTRUCTION WORKERS DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	1.43E-07	6.03E-09	1.70E-06	7.11E-08	1.71E-07	7.23E-09	0.0001839706	0.0000171143	0.0000008943	7.2342186E-11
Benzo(a)anthracene	3.93E-08	1.66E-09	4.67E-07	1.98E-08	4.71E-08	1.99E-09	0.0000506786	0.0000047143	0.0000000357	1.9927313E-11
Benzo(b)fluoranthene	1.69E-07	7.14E-09	2.01E-06	8.49E-08	2.03E-07	8.56E-09	0.0002177643	0.0000202571	0.0000001482	8.5627062E-11
Benzo(k)fluoranthene	1.69E-07	7.14E-09	2.01E-06	8.49E-08	2.03E-07	8.56E-09	0.0002177643	0.0000202571	0.0000000699	8.5627062E-11
Benzo(g,h,i)perylene	3.93E-08	1.66E-09	4.67E-07	1.98E-08	4.71E-08	1.99E-09	0.0000506786	0.0000047143	0.0000000054	1.9927313E-11
Chrysene	2.77E-07	1.17E-08	3.30E-06	1.39E-07	3.32E-07	1.40E-08	0.0003572071	0.0000332286	0.0000000069	1.4045737E-10
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	3.93E-08	1.66E-09	4.67E-07	1.98E-08	4.71E-08	1.99E-09	0.0001013571	0.0000094286	0	9.9636567E-12
Fluorene	6.95E-08	2.94E-09	8.27E-07	3.50E-08	8.34E-08	3.53E-09	0.0000896857	0.0000083429	0	3.5265306E-11
Anthracene	6.10E-07	2.58E-08	7.26E-06	3.07E-07	7.32E-07	3.10E-08	0.0007872071	0.0000732286	0	0.0000000003
Phenanthrene	6.10E-07	2.58E-08	7.26E-06	3.07E-07	7.32E-07	3.10E-08	0.0007872071	0.0000732286	0	0.0000000003
Fluoranthene	3.93E-08	1.66E-09	4.67E-07	1.98E-08	4.71E-08	1.99E-09	0.0000506786	0.0000047143	0	1.9927313E-11
Pyrene	4.77E-07	2.02E-08	5.68E-06	2.40E-07	5.72E-07	2.42E-08	0.0006152071	0.0000572286	0	0.0000000002
Acenaphthene	6.24E-08	2.64E-09	7.42E-07	3.14E-08	7.49E-08	3.16E-09	0.0000804714	0.0000074857	0	3.1642158E-11
Pesticides										
Aldrin	9.83E-09	4.16E-10	1.17E-07	4.95E-09	1.18E-08	4.99E-10	0.0042283333	0.0003933333	0.0000000912	1.4963601E-14
Dieldrin	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0001535714	0.0000142857	0.0000000057	1.5096450E-15
Heptachlor	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0000255952	0.000002381	0.0000000015	9.0578697E-15
Heptachlor Epoxide	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0005906593	0.0000549451	0.000000003	3.9250769E-16
DDT*	4.62E-09	1.95E-10	5.50E-08	2.32E-09	5.54E-09	2.34E-10	0.0001191714	0.0000110857	0.0000000009	1.1714845E-13
Endosulfan I	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0001535714	0.0000142857	0	1.5096450E-15
Endosulfan II	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0001535714	0.0000142857	0	1.5096450E-15
Endosulfan sulfate	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0001535714	0.0000142857	0	1.5096450E-15
Alpha-BHC	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0000255952	0.000002381	0.000000002	9.0578697E-15
Beta-BHC	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0000255952	0.000002381	0.0000000006	9.0578697E-15
Gamma-BHC	5.95E-10	2.52E-11	7.08E-09	2.99E-10	7.14E-10	3.02E-11	0.0000255952	0.000002381	0.0000000004	9.0578697E-15
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors	1.67E-08	7.07E-10	1.99E-07	8.41E-09	0.00E+00	0.00E+00	0.0021561429	0	0.0000000702	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	6.00E-08	2.54E-09	7.14E-07	3.02E-08	7.94E-08	3.35E-09	0.0000387	0.0000039683	0	6.7095331E-11
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE H-10
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND INHALATION

SCENARIO: PHASE II EXPOSED CONSTRUCTION WORKERS DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	3.73E-06	1.58E-07	0.00E+00	0.00E+00	1.34E-06	5.68E-08	0.0037301587	0.0013428571	0.0000002365	5.6762650E-11
Lead	1.37E-05	5.79E-07	0.00E+00	0.00E+00	1.64E-05	6.94E-07	0.0273809524	0.0328571429	0	0.0000000003
Mercury	1.98E-08	8.39E-10	0.00E+00	0.00E+00	7.14E-09	3.02E-10	0.0000661376	0.0000238095	0	9.0578697E-14
Zinc	4.68E-05	1.98E-06	0.00E+00	0.00E+00	1.69E-05	7.13E-07	0.0002229781	0.0016857143	0	0.0000000071
Copper	9.92E-06	4.19E-07	0.00E+00	0.00E+00	3.57E-06	1.51E-07	0.0002681253	0.0003571429	0	0.0000000015
Silver	5.16E-07	2.18E-08	0.00E+00	0.00E+00	1.86E-07	7.85E-09	0.0001719577	0.0000619048	0	2.3550461E-11
Thallium	3.85E-06	1.63E-07	0.00E+00	0.00E+00	1.39E-06	5.86E-08	0.0549886621	0.0197959184	0	4.1001957E-12
TOTAL SITE HAZARD INDEX								0.1552569628		
TOTAL SITE CANCER RISK										1.58E-06

TABLE B-11
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE III EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-09	7.54E-11	0	0.0000001783	0	7.5356444E-13
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	2.08E-11	0	0.0000000491	0	2.0757618E-13
Benzo(b)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.11E-09	8.92E-11	0	0.000000211	0	8.9194856E-13
Benzo(k)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.11E-09	8.92E-11	0	0.000000211	0	8.9194856E-13
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	2.08E-11	0	0.0000000491	0	2.0757618E-13
Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.46E-09	1.46E-10	0	0.0000003461	0	1.4630976E-12
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	2.08E-11	0	0.0000000982	0	1.0378809E-13
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.69E-10	3.67E-11	0	0.0000000869	0	3.6734694E-13
Anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-09	3.22E-10	0	0.0000007628	0	3.2243500E-12
Phenanthrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.63E-09	3.22E-10	0	0.0000007628	0	3.2243500E-12
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.91E-10	2.08E-11	0	0.0000000491	0	2.0757618E-13
Pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.96E-09	2.52E-10	0	0.0000005961	0	2.5198490E-12
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.80E-10	3.30E-11	0	0.000000078	0	3.2960581E-13
Pesticides										
Aldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.23E-10	5.20E-12	0	0.0000040972	0	1.5587084E-16
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000001488	0	1.5725468E-17
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000000248	0	9.4352810E-17
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000005723	0	4.0886218E-18
DDTR	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.77E-11	2.44E-12	0	0.0000001155	0	1.2202963E-15
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000001488	0	1.5725468E-17
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000001488	0	1.5725468E-17
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000001488	0	1.5725468E-17
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000000248	0	9.4352810E-17
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000000248	0	9.4352810E-17
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-12	3.15E-13	0	0.0000000248	0	9.4352810E-17
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.27E-10	3.49E-11	0	0.0000000413	0	6.9890970E-13
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-11
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE III EXPOSED PEDESTRIAN COMMUTER DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.40E-08	5.91E-10	0	0.0000139881	0	5.9127761E-13
Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-07	7.23E-09	0	0.0003422619	0	3.6168577E-12
Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.44E-11	3.15E-12	0	0.000000240	0	9.4352810E-16
Zinc	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.76E-07	7.42E-09	0	0.0000175595	0	7.4224210E-11
Copper	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.72E-08	1.57E-09	0	0.0000037202	0	1.5725468E-11
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.93E-09	8.18E-11	0	0.0000006448	0	2.4531731E-13
Thallium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-08	6.10E-10	0	0.0002082075	0	4.2710372E-14
TOTAL SITE HAZARD INDEX								0.0005936295		
TOTAL SITE CANCER RISK										1.0954009E-10

TABLE B-12
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE III EXPOSED OFF SITE CHILDREN DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.66E-08	7.03E-10	0	0.0000016639	0	7.0332681E-12
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	1.94E-10	0	0.0000004583	0	1.9373777E-12
Benzo(b)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-08	8.32E-10	0	0.0000019694	0	8.3248532E-12
Benzo(k)fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.97E-08	8.32E-10	0	0.0000019694	0	8.3248532E-12
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	1.94E-10	0	0.0000004583	0	1.9373777E-12
Chrysene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.23E-08	1.37E-09	0	0.0000032306	0	1.3655577E-11
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(s)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	1.94E-10	0	0.0000009167	0	9.5868885E-13
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.11E-09	3.43E-10	0	0.0000008111	0	3.4285714E-12
Anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.12E-08	3.01E-09	0	0.0000071194	0	3.0093933E-11
Phenanthrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.12E-08	3.01E-09	0	0.0000071194	0	3.0093933E-11
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.58E-09	1.94E-10	0	0.0000004583	0	1.9373777E-12
Pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.56E-08	2.35E-09	0	0.0000055639	0	2.3518591E-11
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.28E-09	3.08E-10	0	0.0000007278	0	3.0763209E-12
Pesticides										
Aldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.15E-09	4.85E-11	0	0.0000382407	0	1.4547945E-15
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000013889	0	1.4677104E-16
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000002315	0	8.8062622E-16
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000053419	0	3.8160470E-17
DDTR*	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.39E-10	2.28E-11	0	0.0000010778	0	1.1389432E-14
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000013889	0	1.4677104E-16
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000013889	0	1.4677104E-16
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000013889	0	1.4677104E-16
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000002315	0	8.8062622E-16
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000002315	0	8.8062622E-16
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-11	2.94E-12	0	0.0000002315	0	8.8062622E-16
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.74E-08	7.34E-10	0	0.0000008681	0	1.4677104E-11
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-12
AVERAGE DAILY DOSE AND CALCULATED RISK FROM INHALATION

SCENARIO: PHASE III EXPOSED OFF SITE CHILDREN DURING BUILDING CONSTRUCTION

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.31E-07	5.52E-09	0	0.0001305556	0	5.5185910E-12
Lead	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.60E-06	6.75E-08	0	0.0031944444	0	3.3757339E-11
Mercury	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.94E-10	2.94E-11	0	0.0000023148	0	8.8062622E-15
Zinc	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.64E-06	6.93E-08	0	0.0001638889	0	0.0000000007
Copper	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.47E-07	1.47E-08	0	0.0000347222	0	1.4677104E-10
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.81E-08	7.63E-10	0	0.0000060185	0	2.2896282E-12
Thallium	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.35E-07	5.69E-09	0	0.0019246032	0	3.9863014E-13
TOTAL SITE HAZARD INDEX								0.0055410242		
TOTAL SITE CANCER RISK										1.03E-09

TABLE B-13
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND DERMAL CONTACT

SCENARIO: POST CONSTRUCTION -- DAYCARE CHILDREN IN PROPOSED PLAYGROUND

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Carcinogenic PAH Compounds										
Benzo(a)pyrene	5.18E-07	3.70E-08	8.89E-07	6.35E-08	0.00E+00	0.00E+00	0.0001406712	0	0.0000011555	0
Benzo(a)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Benzo(b)fluoranthene	5.18E-07	3.70E-08	8.89E-07	6.35E-08	0.00E+00	0.00E+00	0.0001406712	0	0.0000001618	0
Benzo(k)fluoranthene	5.18E-07	3.70E-08	8.89E-07	6.35E-08	0.00E+00	0.00E+00	0.0001406712	0	0.0000000763	0
Benzo(g,h,i)perylene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chrysene	8.05E-07	5.75E-08	1.38E-06	9.88E-08	0.00E+00	0.00E+00	0.0002188219	0	0.0000000072	0
Dibenzo(a,h)anthracene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Indeno(1,2,3-c,d)pyrene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Noncarcinogenic PAH Compounds										
Naphthalene(a)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Fluorene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Anthracene	1.75E-06	1.25E-07	3.01E-06	2.15E-07	0.00E+00	0.00E+00	0.0004767192	0	0	0
Phenanthrene	1.75E-06	1.25E-07	3.01E-06	2.15E-07	0.00E+00	0.00E+00	0.0004767192	0	0	0
Fluoranthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Pyrene	1.61E-06	1.15E-07	2.77E-06	1.98E-07	0.00E+00	0.00E+00	0.0004376438	0	0	0
Acenaphthene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Pesticides										
Aldrin	3.74E-08	2.67E-09	6.42E-08	4.59E-09	0.00E+00	0.00E+00	0.0033865297	0	0.0000001234	0
Dieldrin	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Heptachlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Heptachlor Epoxide	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
DDTR*	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan I	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan II	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Endosulfan sulfate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Alpha-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Beta-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Gamma-BHC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Methoxychlor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Total Arochlors				0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Phthalate Esters										
Bis(2-ethylhexyl) phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-octyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Di-N-butyl phthalate	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Volatile Organic Compounds										
Benzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chlorobenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0

TABLE B-13
AVERAGE DAILY DOSE AND CALCULATED RISK FROM SOIL INGESTION AND DERMAL CONTACT

SCENARIO: POST CONSTRUCTION -- DAYCARE CHILDREN IN PROPOSED PLAYGROUND

CONTAMINANT	ADD(CHRONIC) INGESTION (MG/KG/DAY)	ADD(LIFE) INGESTION (MG/KG/DAY)	ADD(CHRONIC) DERMAL (MG/KG/DAY)	ADD(LIFE) DERMAL (MG/KG/DAY)	ADD(CHRONIC) INHALATION (MG/KG/DAY)	ADD(LIFE) INHALATION (MG/KG/DAY)	ORAL/DERMAL HAZARD INDEX	INHALATION HAZARD INDEX	ORAL/DERMAL CARCINOGENIC RISK	INHALATION CARCINOGENIC RISK
Acetone	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Methylene Chloride	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,2 Dichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Chloroform	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
1,1,2,2 Tetrachloroethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Toluene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Ethylbenzene	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Trichlorofluoromethane	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00				
Xylenes	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Metals										
Arsenic	4.60E-06	3.29E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0046027397	0	0.0000004932	0
Lead	2.16E-05	1.54E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0431506849	0	0	0
Mercury	4.51E-07	3.22E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0015022831	0	0	0
Zinc	6.81E-05	4.86E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0003242009	0	0	0
Copper	5.75E-05	4.11E-06	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0015549796	0	0	0
Silver	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0	0	0	0
Thallium	8.63E-06	6.16E-07	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.1232876712	0	0	0
TOTAL SITE HAZARD INDEX								0.179841007		
TOTAL SITE CANCER RISK										2.02E-06

TABLE B-14

SUMMARY OF COEFFICIENTS

CONTAMINANT	ORAL ABSORPTION FACTOR	DERMAL ABSORPTION FACTOR	INHALATION ABSORPTION FACTOR	ORAL CPF (MG/KG/DAY)-1	INHALATION CPF (MG/KG/DAY)-1	ORAL RFD/AIC (MG/KG/DAY)	INHALATION RFD/AIC (MG/KG/DAY)
Carcinogenic PAH Compounds							
Benzo(a)pyrene	0.3	0.05	1	11.500	6.100	0.010	0.010
Benzo(a)anthracene	0.3	0.05	1	1.668	0.884	0.010	0.010
Benzo(b)fluoranthene	0.3	0.05	1	1.610	0.854	0.010	0.010
Benzo(k)fluoranthene	0.3	0.05	1	0.759	0.403	0.010	0.010
Benzo(g,h,i)perylene	0.3	0.05	1	0.253	0.134	0.010	0.010
Chrysene	0.3	0.05	1	0.046	0.024	0.010	0.010
Dibenzo(a,h)anthracene	0.3	0.05	1	12.765	6.771	0.010	0.010
Indeno(1,2,3-c,d)pyrene	0.3	0.05	1	2.668	1.415	0.010	0.010
Noncarcinogenic PAH Compounds							
Naphthalene(s)	0.3	0.05	1	NA	NA	0.005	0.005
Fluorene	0.3	0.05	1	NA	NA	0.010	0.010
Anthracene	0.3	0.05	1	NA	NA	0.010	0.010
Phenanthrene	0.3	0.05	1	NA	NA	0.010	0.010
Fluoranthene	0.3	0.05	1	NA	NA	0.010	0.010
Pyrene	0.3	0.05	1	NA	NA	0.010	0.010
Acenaphthene	0.3	0.05	1	NA	NA	0.010	0.010
Pesticides							
Aldrin	0.3	0.05	1	17.000	17.000	3.000E-05	3.000E-05
Dieldrin	0.3	0.05	1	16.000	16.000	5.000E-05	5.000E-05
Heptachlor	0.3	0.05	1	4.500	4.500	3.000E-04	3.000E-04
Heptachlor Epoxide	0.3	0.05	1	9.100	9.100	1.300E-05	1.300E-05
DDTR*	0.3	0.05	1	0.340	0.340	5.000E-04	5.000E-04
Endosulfan I	0.3	0.05	1	NA	NA	5.000E-05	5.000E-05
Endosulfan II	0.3	0.05	1	NA	NA	5.000E-05	5.000E-05
Endosulfan sulfate	0.3	0.05	1	NA	NA	5.000E-05	5.000E-05
Alpha-BHC	0.3	0.05	1	6.300	6.300	3.000E-04	3.000E-04
Beta-BHC	0.3	0.05	1	1.800	1.800	3.000E-04	3.000E-04
Gamma-BHC	0.3	0.05	1	1.300	1.300	3.000E-04	3.000E-04
Methyoxychlor	0.3	0.05	1	NA	NA	0.050	0.050
Total Arochlors				7.700	7.700	1.000E-04	1.000E-04
Phthalate Esters							
Bis(2-ethylhexyl) phthalate	0.3	0.05	1	0.014	0.014	0.02	0.02
Di-N-octyl phthalate	0.3	0.05	1	NA	NA	0.02	0.02
Di-N-butyl phthalate	0.3	0.05	1	NA	NA	0.1	0.1
Volatile Organic Compounds							
Benzene	1	0.5	1	0.052	0.026	7.000E-04	7.000E-04
Chlorobenzene	1	0.5	1	NA	NA	0.005	0.005

TABLE 8-14

SUMMARY OF COEFFICIENTS

CONTAMINANT	ORAL ABSORPTION FACTOR	DERMAL ABSORPTION FACTOR	INHALATION ABSORPTION FACTOR	ORAL CPF (MG/KG/DAY)-1	INHALATION CPF (MG/KG/DAY)-1	ORAL RFD/AIC (MG/KG/DAY)	INHALATION RFD/AIC (MG/KG/DAY)
Acetone	1	0.5	1	NA	NA	NA	NA
Methylene Chloride	1	0.5	1	0.008	0.014	0.060	0.060
1,2 Dichloroethane	1	0.5	1	0.091	0.035	0.074	0.074
Chloroform	1	0.5	1	0.006	0.081	0.010	0.010
Trichloroethane	1	0.5	1	0.057	0.057	0.004	0.004
1,1,2,2 Tetrachloroethane	1	0.5	1	0.200	0.200	NA	NA
Toluene	1	0.5	1	NA	NA	0.300	1.500
Ethylbenzene	1	0.5	1	NA	NA	0.100	0.100
Trichlorofluoromethane	1	0.5	1	NA	NA	NA	NA
Xylenes	1	0.5	1	NA	NA	2.000	2.000
Metals							
Arsenic	1	0	1	1.500	5.000	0.001	0.001
Lead	0.3	0	1	NA	NA	5.000E-04	5.000E-04
Mercury	1	0	1	NA	NA	3.000E-04	3.000E-04
Zinc	1	0	1	NA	NA	0.210	0.010
Copper	1	0	1	NA	NA	0.037	0.010
Silver	1	0	1	NA	NA	0.003	0.003
Thallium	1	0	1	NA	NA	7.000E-05	7.000E-05

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